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SOME PROBLEMS OF VARICOSE VEINS.

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VARICOSE VEINS are as stubborn as mules and, like mules, do their best to resist treatment, kindly or otherwise. It is quite true that much more hope can be given to sufferers from this complaint nowadays and that the prospects of cure are better than ever before. There is no reason, because of this, to sit back contentedly and say: "There's another disease mastered." Far from it. Many varicosities recur after treatment; in other cases there is not the lessening in pain, swelling, dermatitis, ulceration *et cetera* that would be expected with obliteration of the veins. Moreover, uncertainty exists with regard to ætiology, anatomy, pathology and treatment. So the subject cannot be dismissed airily.

It is not my object in this article to go into all the problems on the subject, but rather to deal with the practical approach to cases of varicose veins in the light of more recent knowledge.

Anatomy.

There are superficial, deep and communicating veins; there are long and short saphenous veins (also called internal and external, greater and lesser), which collect (through many tributaries) most of the blood from the superficial areas of the limb; the flow of blood is directed by valves from the superficial to the deep veins. All this is known, as are the sites of the main communicating veins in the leg and the thigh. This knowledge has led to Figure I, which puts it all very clearly. It should be remembered, however, that it is a diagrammatic and not a full representation of the venous system of the lower limb. The large recurrences seen after misguided solitary ligation and division of the long saphenous vein in mid-thigh or leg show that there are many other veins in the limb capable of equalling or even of excelling the main saphenous trunk in capacity and guilt.

Near the termination of the long saphenous vein all manner of variety is found, with the three superficial tributaries and the medial and lateral femoral cutaneous veins and the long saphenous vein itself, which may be single or double or in the form of a loop.

In the lower part of the thigh another bewildering array of tributaries and junctions is found between the communicating veins of the middle part of Hunter's canal and the *adductor magnus* hiatus regions, the subsartorial veins, the genicular plexus and the saphenous veins. (See phlebograms, Figures II and III.) Figure II shows a communicating vein entering the femoral vein in the middle of the Hunter canal region, with some small tributaries. Figure III is a picture taken after division of the long saphenous vein at the knee and injection of opaque material into its distal end. It shows, in addition to the deep veins of the upper part of the leg and popliteal regions, a very complex maze of veins in the region of the knee. Some of these are quite large and different from the lace-like superficial veins in the upper corner. These anatomical points have bearing on treatment and will be referred to again.

Ætiology.

Varicose veins can appear, that is, become apparent, at any age from fourteen years onwards (maybe someone knows of an earlier age). Childbirth, long standing, garters, hormones, congenital defects all play a part. (So far vitamins and psychosomatic tendencies are exempt from blame.) What part hormones play and which are the hormones are not known. The theory is advanced because varicose veins often manifest themselves at important "hormonal seasons"—puberty, childbirth, the menopause. The argument is unconvincing. Main causes seem

to be (*a*) developmental or congenital and (*b*) physical, that is, pressure effects. Both have some underlying factor, namely, valvular deficiency.

There are two main types of varicose veins: (i) the true, bulging, comparatively thin-walled varicosities seen in older people, particularly in women after pregnancies, and (ii) the thick-walled tortuosities commonly seen first in young people of about twenty years. There seems to be an essential difference in their ætiology. The former were once normal veins which have been made varicose by unusual severe pressure effects applied suddenly, that is, for a comparatively short time; the latter can be said never to have been normal. They are the result of a long-continued steady pressure of lesser severity, brought about by developmental valvular defects, and present from the time the child stands up, or maybe earlier. The familial tendency in varicose veins is known to be strong. The underlying factor in all cases is the same, namely, a response to abnormal pressures. It seems unlikely that a sudden flood of hormones at puberty can pick out the saphenous system and transform a normal vein into the very thick-walled structures seen commonly in young people. However, it may be that the power of hormones is underestimated and that a combination of factors is at work. Proof of these theories is difficult, but the fact remains that no treatment based on hormone therapy has been accepted as effective, and our approach to treatment is entirely directed towards overcoming physical defects.

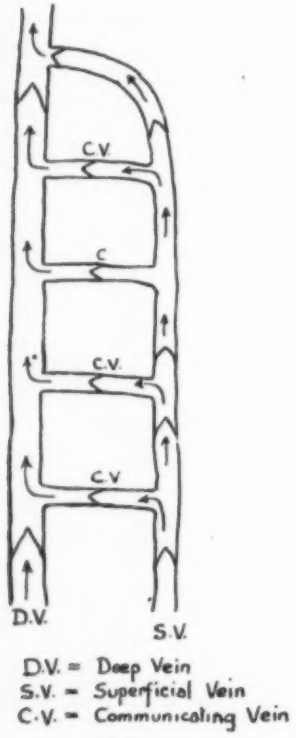

FIGURE I.

Treatment.

Present-day operative measures in the treatment of varicose veins still give rise to criticism and dissatisfaction. Certainly some very poor results are seen. This is unfortunate, for with thought, painstaking care and attention to detail excellent results can be obtained in a majority of cases. While admitting that some difference of opinion exists as to just how extensive operative procedures should be to ensure a cure, I believe that most bad results are the result of bad technique. This point will be discussed later, but first some of the more common causes of failure will be dealt with.

Use of the Word "Ligation".

"Saphenous ligation" is a commonly used phrase which is heard and seen everywhere, even on operation lists. Most surgeons, when they say saphenous ligation, mean saphenous ligation and section, and this is the operation they do; but saphenous ligation is often done in the belief that it is the usual operation and that ligation of a vein permanently occludes it. This is quite wrong.

A young woman developed varicose veins with her first pregnancy and later had the "vein tied in five places". On examination six months after operation the only effect was five tiny healed scars in the course of the veins which were said to be, if anything, bigger than before. No precise details were available beyond the above history, and no details, of course, as to what material was used for ligation.

This case is similar to others, in which a vein runs right through the line of the scar of a past operation. More positive evidence is afforded by the following case.

A male patient, M., aged thirty-three years, had varicose veins for thirteen years. About 1932 a bunch of veins was excised just below the knee; in 1939 many injections were given; in 1942 an operation was performed on the vein near the groin. After the last procedure the condition improved for a short time and then recurred. On examination the scars of previous operations were present (*a*) in the thigh and (*b*) below the knee. In the thigh the incision had been too low, that is, two and a half inches below the *fossa ovalis*, and the long saphenous vein ran right through the scar, being easily palpable. Incidentally, at the knee the site of the earlier excision had been neatly side-tracked by two tributaries each as big as the long saphenous vein itself. One ran in front and the other behind the scar and united with the main vein above. There were large varicosities in thigh and leg and the Trendelenburg sign was present. Operation confirmed the clinical findings. The superficial tributaries at the upper end of the long saphenous vein were large and intact. Medial and lateral femoral cutaneous veins were also seen and all these vessels were divided. Exploration at the site of the scar showed the long saphenous vein to be intact, but white and thickened for about half an inch, deep to the scar. When opened, it was found to be filled with fibrous tissue, but had become recanalized in two places. The section (Figures IV and V) shows this condition clearly and microscopic examination shows the vein to be occluded by dense fibrous tissue with cholesterol clefts. Recanalization is through one large and several smaller channels, lined by endothelium.

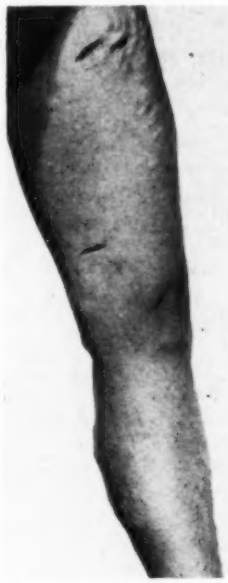

FIGURE VI.

At the original operation the vein had been ligated, presumably with catgut. The vein had become recanalized and the condition had recurred. The conclusion can safely be drawn that ligation with catgut is quite unreliable. Whether the use of non-absorbable material would be satisfactory is uncertain. What is certain is that use of the term saphenous ligation and the operation described by it should be dropped and saphenous section substituted.

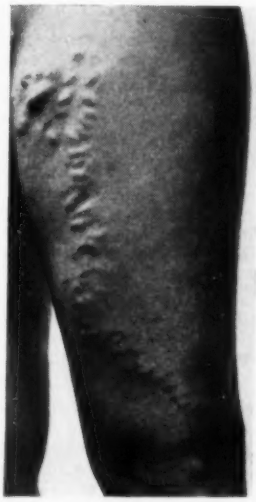

FIGURE VII.

Site of Section.

No matter what are the situation and extent of the varicosities, if operation is decided upon, the long saphenous vein must be divided at its extreme upper end and its tributaries in this region must be divided also. This might be termed the "basic operation". It forms the starting point of all operations on varicose veins in the lower extremities. This applies even if only the short saphenous vein is obviously varicose, for the communication between the long and short veins is quite free.

There is still a strong tendency to divide the vein two or three inches below the *fossa ovalis* in spite of the fact that this—the original Trendelenburg operation—was tried and discarded many years ago. There are reasons for this being done: (*a*) It is easier. (*b*) It is thought by many that division of the vein at its upper end is not really necessary and that division of superficial tributaries is a theoretical refinement. (*c*) There is a belief that there is less risk of thrombosis and embolism in the lower operation. (*d*) A very popular idea persists that the procedure is just a "minor op.". These points will be dealt with in more detail.

That the operation is easier is true, but this is no reason for incorrect treatment. Lower down, the long saphenous and femoral veins are well apart, and damage to the femoral vein, which can and does occur in the high operation, is unlikely to occur. Moreover, other difficulties, mentioned later, are not encountered. The difficulties of the operation are increased by the cult of the small incision. The operator who through a small incision inserts a pair of artery forceps and opens and closes them in a hopeful grope for a vessel intimately associated with the femoral vein and having thin-walled tributaries will surely rue the day. A generous incision and a good look are the best insurance against trouble. (An excellent ally is a Watson-Cheyne dissector.)

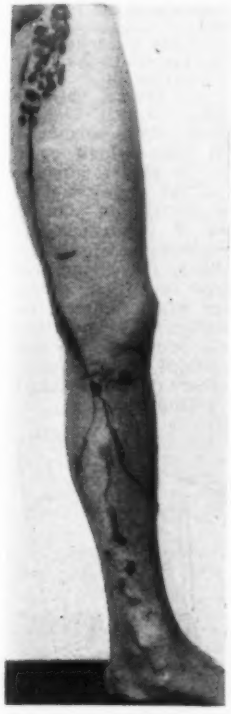

FIGURE VIII.

That division of the vein at its upper end is not really necessary and is a refinement, is a delusion and an excuse for dodging the most tedious and difficult part of the operation. Proof of the necessity of dividing the tributaries is afforded by the following cases and their pictures.

A patient, W., had had varicose veins for six years. He was submitted to operation in February, 1942, as an out-patient. Recurrences started in six months. Figures VI and VII show: (i) The sites of operations in the thigh (note that the upper one is rather low). (ii) Massive recurrence in the region of and particularly to the outer side of the upper scar. (iii) A large vein running down the centre of the thigh. (iv) Large bulky veins in the calf.

At subsequent operation a large saphenous varix with a large lateral tributary running into it was found. The pressure transmitted through this undivided tributary had caused a new varicose chain in the thigh, lateral to the original long saphenous vein which had apparently been obliterated, and large recurrences in the leg extending almost to the ankle. The side-tracking of the section in the knee region had been a simple matter, as it usually is in the absence of properly performed "basic" operation.

A patient, B., was operated on in 1941 for varicose veins. According to notes made by the surgeon, the saphenous vein was found, three large tributaries were ligated at the saphenous opening and a retrograde injection was given. A vein was secured also just above and below the knee. A year later some injections were given into the calf. Varicosities gradually recurred, and Figures VIII and IX show the condition present in 1945. There were massive varicosities lateral to, and below, the scar of the original operation which is properly high (compare with previous case), recanalization of the long saphenous with the usual side-tracking of the lower thigh scar, and large bunches of varicosities in the leg and calf extending right to the ankle. At subsequent operation a large lateral tributary was found.

What had been the fault at the original operation is difficult to decide. Either the word "ligated" meant what it said, in which case recurrence could have taken place by recanalization of the "ligated" vein, or a tributary, that at the time was small and so overlooked, had enlarged and caused recurrence. It is worthy of note that even with a careful operation, as in this case, recurrence can take place through a comparatively small error in technique.

FIGURE IX.

A patient, Br., had had varicose veins for fifteen years. He had had thirty injections in the last four years and operation thirteen months prior to being seen. He stated that he had had no relief from operation. The picture of the left limb (Figure X) shows the condition present, that is, a very low incision with recurrence round the medial side of the scar. The veins were located by palpation and their position was marked. The picture therefore represents a pre-operative estimate of venous arrangement. There were very large varicosities in the rest of the limb. At operation the condition shown in Figure XI was found. There was the piece of the upper end of the long saphenous vein above the original point of section with three large tributaries running into its upper end. At its lower end were several small tributaries, and on the medial side a large vein (v) establishing continuity of the long saphenous vein. This was either a tributary enlarged by pressure, or one side of a loop of the long saphenous, missed at the original operation. Removal of the whole of the upper stump and division of all tributaries and retrograde injection into (v) were carried out. Dissection in the region of the scar was difficult, as was identification of (v) as the main vessel responsible for the recurrence.

FIGURE X.

Fear of thrombosis leading to pulmonary embolism, the third reason mentioned, is ever present. Fortunately it seldom happens. A massive embolus is not likely to come from the superficial system. It is now known that most cases of pulmonary embolism follow thrombosis in the deep venous system of the lower limb and that fatal cases are caused by detachment of a long floating thrombus in the femoral and popliteal veins. Thrombus formation proximal to the site of ligation and section must be admitted as a probability. In the case of operation as close as possible to the femoral vein, only a very small area is available for thrombus formation. (See Figures XII and XIII.) Even should this occur here, it is unlikely to propagate as a large floating thrombus into the femoral vein, where the stream is more swiftly moving, provided the patient moves about freely after operation. Small emboli might break off—and sometimes do—and lodge in the lung without very serious consequences. (Included in

these, however, is an acute anxiety state on the part of the surgeon.)

The lower the operation is performed, the larger the area for thrombosis in the stagnant upper limb of the long saphenous vein and the larger the possible embolus with correspondingly more serious affects. In the very low operation, that is, in the middle or lower part of the thigh, stagnation in the upper limb of the vein is less likely to occur owing to the entrance of tributaries. In any case the likelihood of fatal embolus from the superficial system is not great, provided, of course, as mentioned before, the patient moves about freely after operation. This also reduces the possibility of deep vein thrombosis. A greater reason for fear lies in the fact that damage to the femoral vein is more likely in the higher operation. This vein has been divided in mistake for the saphenous and it has been damaged during operation, being incised, torn or included in the clamp used to control hæmorrhage. These risks call for greater care during the operation, not for the performance of the lower operation.

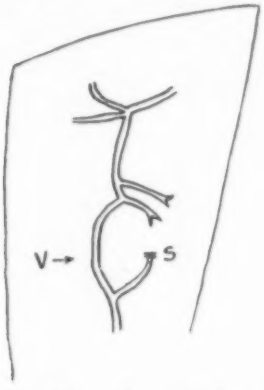

FIGURE XI.

As long as the operation is regarded as a minor one (the fourth reason mentioned above), so long will minor procedures be carried out. Division of a palpable or visible vein or both in the middle part of the thigh is a minor operation. Radical operation for varicose veins, including the "basic operation" and such secondary divisions and excisions as may be necessary in other parts of the limbs, is not a minor procedure. Great care is needed in the region of the *fossa ovalis*, for the long saphenous vein may be, and the tributaries always are, thin walled, and tearing with free hæmorrhage can easily occur. Difficulties are also encountered because of fat, variation in anatomical arrangements of veins, and, in cases of ulcers and dermatitis, from enlarged lymph glands and lymphatic channels and fibrosis round the main vein and its tributaries. A good light, a good exposure, a good assistant, and full surgical asepsis are necessary. Dodd[1] states:

> In closing I would stress the following points: (i) Necessity for complete diagnosis—there are at least seven possibilities. (ii) The need to upgrade the condition and operation to a major one.

Obliteration of Superficial Veins.

Failure to obliterate all or a large proportion of varicose veins at the time of operation tends strongly to aid recurrences. Obliteration is done by retrograde injection at the time of operation at each of the sites where section has been necessary. This might be termed "mass obliteration". Some surgeons do not believe in this treatment, but prefer to obliterate the veins by injections given after the operation. The main reason is the fear that the retrograde injection will enter the deep veins and cause thrombosis there. There is no doubt that retrograde injection does enter the deep system. Figure II shows that this occurs, but the poor shadow in the femoral vein suggests that any material entering is diluted and rapidly swept away. In actual practice it is a complication very rarely seen and very rarely reported. Figure XIV is a phlebogram which shows the remarkable ramification of opaque material with retrograde injection, and it is quite certain that sclerosing material causes widespread thrombosis, not only in the large obvious veins, but also in many smaller veins imperceptible by ordinary methods of examination. This is also borne out by the following observation:

FIGURE XII.

FIGURE XIII.

> A patient suffering from varicose veins needed section of both long and short saphenous veins. The long saphenous was divided at the *fossa ovalis* and at the knee; a retrograde injection was given at each place. He was then turned over and the incision was made to expose the short saphenous vein. Small veins in the fat, when cut, exuded dark sticky material—obviously blood mixed with ethamolin. These vessels would, of course, become obliterated by clot.

It is clear that if collateral veins—even very small ones—which could possibly give rise to recurrences are occluded and if at the same time the main saphenous system is blocked throughout its length, the chances of recurrence are reduced to a minimum. When varicosities are left after operation and dealt with by injections, the chances of a successful result are not so good as when "mass obliteration" is achieved. Many veins will be missed, for the whole system shrinks after operation and potentially recurrent channels will not be obvious enough to warrant attention. Moreover, although a successful "take" is more likely after operation than before, there is no certainty that main varicosities will respond to injections.

Varicose Anatomy.

The somewhat strange term, varicose anatomy, is not intended to suggest that anatomy in varicose veins is different from normal anatomy. Rather is it used (*a*) to draw attention to certain anatomical features that are seen often in the treatment of varicose veins and may interfere with the success of treatment if not appreciated, and (*b*) to cover certain fairly common patterns seen when veins become varicose.

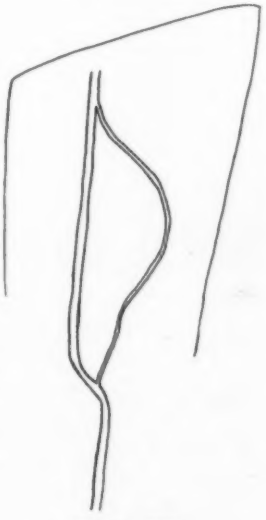

FIGURE XV.

1. At the upper end of the long saphenous vein there are five named tributaries—the medial and lateral femoral cutaneous and the three "superficials", external pudendal, circumflex iliac and epigastric. Each can join with any other, singly or in groups, and with a single or double long saphenous in the same way. To arrive at the number of arrangements, loops, branches and communications possible here we must go back to those delightfully intriguing permutation and combination sums we used to do—or try to do—at school.

2. The long saphenous vein may be double, particularly in the thigh, and may be in the form of a loop, either in the region of the *fossa ovalis*[2] or lower in the thigh. (See Figures XV and XVI.) It will be noticed that in one case (Figure XV) most of the blood was carried by the long saphenous itself and the loop vein was, though quite large, a subsidiary; in the other (Figure XVI) the reverse

condition was present, that is, most of the blood flowed via the loop.

The next case, that of Ba. (see Figures XVII, XVIII and XIX), illustrates the unsatisfactory results that can come from lack of appreciation of these variations.

Ba., a male patient, had had varicose veins for years and was treated by injections, but the condition recurred. In July, 1941, he had an operation, that is, bilateral mid-thigh section of the vein (sites 1 and 2 in figures). This caused little improvement, and six weeks later he had a further operation for vein section (sites 3 and 4). In August, 1942, the condition was as shown in the photograph of clinical estimation of venous arrangement. The loops and double nature of the long saphenous vein can be seen. Only one part of the double vein had been dealt with and the other part had "carried on", and the whole limb was a mass of varicosities. The operations at sites 3 and 4 had made no contribution to a cure. The Trendelenburg sign was strongly positive. At operation the condition shown in Figure XX was found on both sides, in the region of the *fossa ovalis*. The superficial epigastric and pudendal tributaries united to form a common vein and a communication passed down to the medial part of the long saphenous vein. All these vessels were large and thick-walled, and the reflex was quite free.

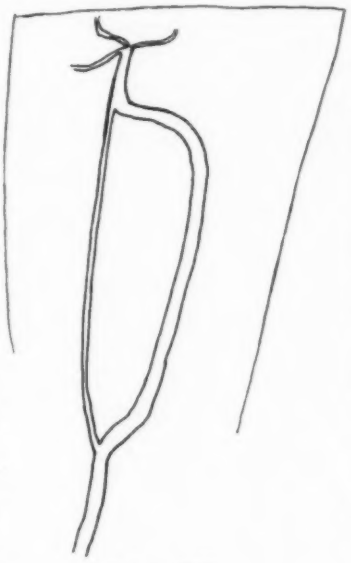

FIGURE XVI.

3. Varicosities may be confined to long or short saphenous veins or both. These are straightforward cases presenting little difficulty. Communication between the veins is free and sometimes quite obvious through large veins.

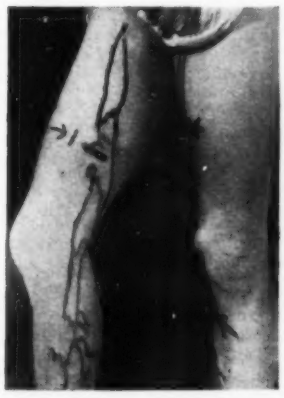

FIGURE XVII.

4. The short saphenous vein may be high. In this case the short vein, instead of ending in the popliteal space, runs up the back of the thigh, turns medially and ends by joining the long saphenous vein in the middle or upper part of the thigh. Another arrangement seen in the thigh is for a large vein coming from the outer side of the leg to run up the outer side of the thigh and cross its anterior aspect diagonally to enter the upper third of the long saphenous vein.

5. In leg varicosities two long veins are often seen, one running up the antero-medial and the other up the postero-medial aspect of the leg and uniting to form the main long saphenous trunk at the knee. Another common tributary is one running up the lateral side of the leg to about its middle and then crossing its front to join the antero-medial tributary mentioned above. Also noted are a vein running across the front of the patella to join the long saphenous vein or its anterior tributary just below the knee and a vein emptying into the upper end of the short saphenous vein. Special care must be taken to deal with this latter vessel.

There is no limit to the bizarre designs made by varicose veins. Some of the strangest and most complicated are seen in recurrent cases.

Further Problems.

While the "basic operation" and "mass obliteration" will give satisfactory results in most cases, there are those in which failure will occur. The satisfactory results are more likely when the communicating veins have effective valves. When reflux occurs through these veins the difficulties are greater, as are the likelihood of failure of relief of symptoms and the recurrence of varicosities. Each of these defectively valved communicating veins can give its own specific Trendelenburg sign and give rise to recurrences if not dealt with, just as the long saphenous vein itself can at the *fossa ovalis*, if not properly treated. To be sure of curing them, each one should be ligated and divided. This may mean several extra incisions and the problem of finding the veins. This will not be so hard if the limb is thin and the gap in the deep fascia can be felt, but may present a real problem in a fat limb from back pressure, ulcer or dermatitis. In any case the operation is growing in magnitude.

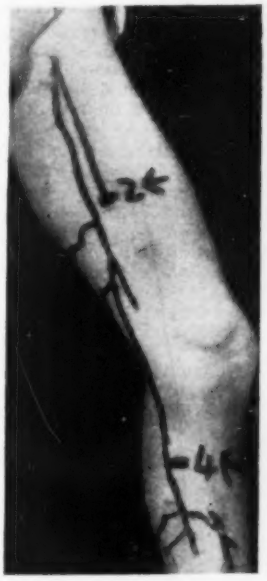

FIGURE XVIII.

Then there is the problem of the veins in the lower part of the thigh and knee region. Free and variable anastomosis occurs here, as previously mentioned. Side-tracking and recurrences are possible here through these veins. So much so that operative procedures have been advocated in this area to eradicate these communications and so to make a cure more certain. Yes, the operation is certainly growing in magnitude. No personal experi-

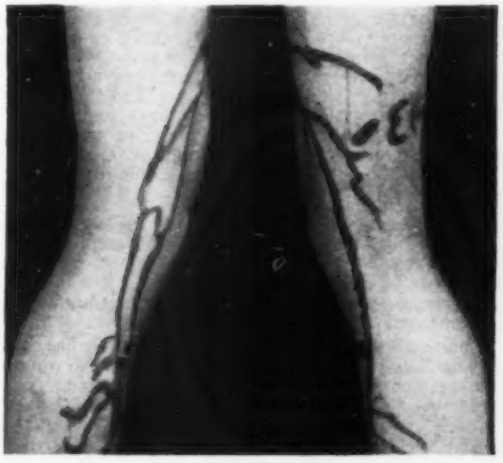

FIGURE XIX.

ences are available for further opinion or comment on this procedure.

Excision of large bunches of veins is often advisable and necessary, particularly when they are associated with underlying defectively valved communicating veins—these two birds can be killed with the one stone. This operation is useless unless combined with the "basic operation", as has been shown so often in the past.

Compensatory veins, that is, enlarged superficial veins in a patient with a past history of deep vein thrombosis, present a problem. The accepted teaching has always been that they should not be touched, as unfortunate results have followed their obliteration. I have, however, seen a

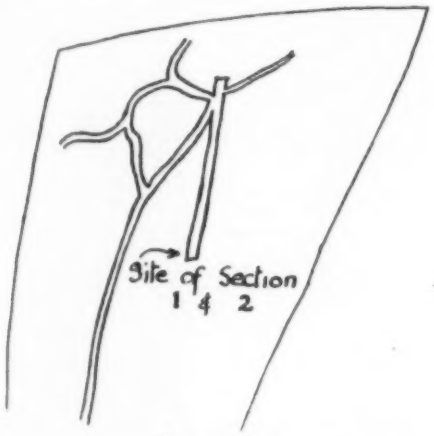

FIGURE XX.

patient with obliteration of the deep veins and a history of earlier femoral thrombosis, who had a thrombophlebitis of the whole length of the long saphenous vein without ill effects. This problem is unsolved and conservatism still seems the safest policy. There are others which could be discussed, but enough has been said to enable certain conclusions to be drawn.

Conclusions.

There is still a great deal we do not know about varicose veins and their treatment. So in seeking cure in a case we must stick grimly to what we do know. When surgery is considered necessary, the "basic operation" is an essential starting point in all cases and no operation should be done without it, while "mass obliteration" is such a useful ally that it should be striven for also in all cases. As to further surgical procedures, each case must be decided on its merits. A cure is possible in the majority of cases and a failure should cause deep thought and self-reproach.

Summary.

1. The anatomy of the veins of the lower extremity is described.

2. The ætiology of varicose veins and "varicose anatomy" are discussed.

3. Operative methods of treatment are described, special emphasis being laid on the need for attention to detail. Some common causes of failure are considered.

Acknowledgements.

I wish to acknowledge the help given by Dr. R. Hoy in the preparation of the phlebograms, and to express my appreciation of Mr. J. W. Brain's photographic studies.

References.

(1) H. Dodd: "Discussion on Varicose Veins", *Proceedings of the Royal Society of Medicine*, February, 1943, page 191.
(2) C. H. W. Lawes: "An Unusual Arrangement of the Long Saphenous Vein", THE MEDICAL JOURNAL OF AUSTRALIA, September 21, 1940, page 261.

DISSECTING ANEURYSM, WITH AN ACCOUNT OF TWENTY-FOUR CASES.

By W. F. EMERY,
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THIS series includes most of the varieties of dissecting aneurysm found, and represents all such cases discovered in 5,600 autopsies conducted at the Royal Adelaide Hospital.

The definition of a dissecting aneurysm has been taken as a dissection of any of the coats of an artery by blood, and as such, includes vessels other than the aorta and coats other than the media. In one of these cases the dissection was confined to the right common iliac artery, and frequently the adventitia was stripped off the media of the aorta, with or without dissection of the latter coat.

Cases of complete rupture of the aorta without dissection are not included, but in some of the cases a partial rupture has occurred and has obviously been followed by dissection.

The history of dissecting aneurysms dates back to the early eighteenth century; an excellent account of the acquisition of knowledge on the subject is given by Shennan,[1] and a more concise account by Flaxman.[2] Shennan has collected and studied 300 authentic cases from the literature up to 1933, and about 200 further cases have been recorded; the aim of this paper is to add to the literature a further 24 cases, occurring from 1920 to 1943.

The incidence as found at autopsy has been variously estimated by different authors at between one in 200 and one in 550; the ratio in these cases is one in 230. The distribution of cases per thousand consecutive autopsies has been as follows: none in the first thousand, one in the second thousand, three in the third thousand, seven in the fourth thousand, seven in the fifth thousand and six in the last six hundred. These figures show an unexplained apparent increase.

Although it is usually stated in textbooks that the lesion rarely occurs under the age of fifty years, Sailer[3] states that no age is exempt and that cases have been reported in a male infant aged fourteen months and in a boy aged ten years; Klotz and Simpson[4] also describe 42 cases occurring in subjects aged under forty years, seven of the subjects being aged between eleven and twenty years. In this series of cases one lesion was in a male aged twenty-seven years and one in a male aged thirty-five years, the remainder being in subjects aged over fifty years. Table I shows that the age incidence has been predominantly in the fifties, sixties and seventies; the age incidence in Holland and Bayley's[5] 19 cases and in Flaxman's[2] 19 cases is included. In Holland and Bayley's cases the incidence seems to have been at a rather lower age than in other series.

Seventeen of the present cases occurred in males and seven in females; this is a ratio of 2·5 to 1, the ratio in much larger series being given as 2 to 1.

Pathology.

The Age of the Dissection.

The age of the dissection, as found at autopsy, may be roughly divided into three classes, as shown by Flaxman:[2] (*a*) recent dissection, (*b*) old "active" dissection, (*c*) old "silent" or "healed" dissection. The majority of the present cases fall into the first class. Of the 24 cases, 17 were of the recent type; in all cases but one death was caused by the dissection. One dissection was of the old "active" type (Case IX)—that is, a recent dissection had been superimposed on an old dissection and in this case had caused death. Six dissections were of the old "silent" type (Cases I, II, V, VI, XII, XIV); three of the subjects eventually died of cerebral hæmorrhage, one of hypostatic pneumonia following cerebral softening, and two of congestive cardiac failure. Thus, in all, 17 deaths out of 24 were caused by the dissection. In Case I of the old "silent"

TABLE I.

Series.	Age (Years).							
	20 to 29	30 to 39	40 to 49	50 to 59	60 to 69	70 to 79	80 to 89	90 to 99
Present series	1	1	0	5	10	5	2	0
Holland and Bayley's cases	2	3	4	4	6	0	0	0
Flaxman's cases	1	1	1	11	4	1	0	0

group the dissection had become endothelialized and was functioning, whereas there was probably no flow of blood through the other five "old" dissections.

The Extent of the Dissection.

The extent of dissection varied from being confined to the ascending aorta to including the whole aorta and common iliac arteries. One dissection was confined to the ascending aorta; five dissections included also a small part of the arch of the aorta; one dissection affected the ascending aorta and proximal half of the arch; one dissection was from the aortic valve to the first few inches of the descending aorta; in three cases the whole thoracic aorta, with a few inches of the abdominal aorta, was dissected; and in five cases the whole length of the aorta was involved, there being continuation to both common iliacs in one case, to the right common iliac in one case, and to both common iliacs and the commencement of the internal iliacs in one. Thus in 16 cases the ascending aorta was dissected, the dissection passing back to the aortic ring in all instances except in Case IX (in which it was 2·5 centimetres from the valve) and in Case XIX (in which it commenced just above the valve). In the remaining eight cases one dissection affected the junction of the arch and descending aorta; two dissections affected the arch of the aorta, one including the first few inches of the descending aorta, and the other passing down to the first few inches of the abdominal aorta; one affected the descending thoracic and a little of the abdominal aorta; three dissections were confined to the abdominal aorta; the remaining dissection was confined to the right common iliac artery. Roughly, the cases fall into the following three groups:

1. Those affecting the ascending aorta, with or without at least a small amount of the arch (16 cases)—this covers three subgroups: (*a*) those affecting the whole aorta (five cases), (*b*) those affecting the thoracic aorta (four cases), and (*c*) those confined to the ascending aorta and first part of the arch (seven cases).
2. Those which affect some part of the arch, but not the ascending aorta, with or without the first few inches of the descending aorta (three cases).
3. Those affecting the remainder of the descending aorta (four cases).

The first group contains all the cases in which hæmopericardium was present; the second group includes most of the cases of hæmothorax, usually left sided; while in the third group external rupture is uncommon.

The extent of dissection round the aorta has been poorly indicated in most cases. Of the sixteen dissections affecting the ascending aorta, in three cases it was noted that the posterior wall was not dissected, in three cases (I, III, XIX) only the anterior wall of the descending aorta was dissected, and in one case (Case I) only the posterior wall was dissected. Of the three dissections confined to the abdominal aorta, one affected the posterior wall and one the anterior wall, and many of the other smaller dissections affected only part of the circumference.

In four cases peripheral arteries as well as the aorta were affected (Cases III, IV, XV and XXIII), the whole extent of the aorta being dissected in three cases and the thoracic aorta in one case; one or both common iliac arteries were affected in three cases; and in two it was noted that the great arteries arising from the aortic arch were affected—namely, the commencements of the innominate, the right common carotid and the left subclavian arteries in Case XV and the first 2·5 centimetres of the right common carotid artery in Case IV.

In some cases a fairly well-defined tear in the intima was found, and this may conveniently be called the "primary" tear, to distinguish it from a "secondary" tear or rupture, either internally (back into the lumen) or externally (into the tissues or cavities outside the aorta). Of six "old" dissections, an old tear was found in five—one just above the valve, one in the ascending aorta, one in the arch (atheromatous aneurysm, Case XII), one in the first part of the descending aorta (Case XIV), and one just above the bifurcation of the aorta. In all, evidence of attempts at healing were found, and in Case XIV the tear had actually become endothelialized, only a depression being left to indicate its position.

Of the 18 cases of recent and recent-on-old dissections, a definite tear was found in ten and a probable tear in one (Case XVIII). Thus in only a little more than one-half of all the cases (15 out of 24) was a definite primary tear (new or old) noticed.

The definition of the recent tears varied considerably, from a small tear in the base of an atheromatous ulcer at the junction of the arch and descending aorta (Case XI) to a clean-cut transverse tear just above the valve, completely encircling the intima and inner part of the media (Case XXIV). Only one other tear (Case XX) was described as "clean-cut", while two were described as "jagged" or "ragged", and two as rectangular or triangular. Six of the recent tears occurred in the ascending aorta, two in the arch, and two at the junction of the arch and descending aorta. Thus in all cases, new and old, eight tears were found in the ascending aorta, three in the arch, two at the junction of the arch and descending aorta, one in the first part of the descending aorta, and one just above the bifurcation. Of the eight tears in the ascending aorta, two were near or just above the aortic valve (Cases I and XVI), five were within five centimetres from the valve, and one was in the ascending aorta more than 2·5 centimetres above the valve. Shennan[1] has found that the majority of all the primary tears occur in the first few centimetres of the ascending aorta.

Three of the tears in the ascending aorta in the present series were transverse, two circular and one rectangular, and in two cases no note was made. Of the two definite tears in the arch, one, just below the innominate, was vertical and 1·5 centimetres long (Case XXII) and one was triangular (Case XXIII). The directions of the tears beyond the arch were not recorded.

The Relation Between Position and Type of Tear and Extent of Dissection.

There appears to be some relation between the position and type of the primary tear and the extent of the dissection. Of the nine cases in which the tear occurred in the ascending aorta or first inch or so of the arch, in one the dissection was confined to the ascending aorta (except the first 2·5 centimetres); in four it extended from the valve to the first inch or so of the arch; in one it was confined to the middle of the arch; in one it was confined to the first few inches of the descending aorta; and in two cases the whole length of the aorta was dissected. Of the five cases in which complete-length dissections were present, in three there were recognizable primary tears.

Of the six cases in which the dissection affected the ascending aorta and the first part of the arch, in only one there was no tear; whereas in the majority of those cases in which the dissection was confined to the descending aorta no primary tear was recognized. In Case XXIII, in which the primary tear was at the distal end of the arch, the medial dissection did not extend far back along the ascending aorta, but was continued in the adventitia to the aortic valve. In Case XIX, however, whereas the tear was at the junction of the arch and the descending aorta, the medial dissection passed back as far as the valve.

Although there are insufficient data to bear it out fully, the general impression is that the larger and deeper the primary tear, the less extensive the dissection.

Atheroma in Relation to the Primary Tear.

The question of atheroma in relation to the primary tear is of some importance. In Cases XI and XII the dissections are obviously atheromatous in nature, the latter being described as an atheromatous aneurysm. In Case XXII the primary tear passed through an atheromatous patch. In all three cases much atheroma, usually of the aorta, was present, increasing from the proximal to the distal end, and associated with many large ulcers in Case XI; but in many of the other cases much atheroma was also present in the distal part of the aorta. Of the remaining 12 cases in which primary tears were present, in four cases there was no mention of atheroma anywhere, in four cases there was no atheroma at the site of the tear, in two cases there was a little atheroma in the region of (though not necessarily through) the tear, hard calcification was present in one case (Case IX), and in one case much atheroma was recorded in the region (Case XIV). Therefore, it seems that in at least 10 of 15 cases there was little or no relation between the primary tear and atheroma.

The "Secondary" Tear.

The next point to consider is the so-called "secondary" tear, usually "external". In one case (Case I) there was evidently an internal reentrance tear, although it had not been sought. Of the seventeen dissections causing death, external rupture in some form or other occurred in all cases; in another recent case (Case XV) no external rupture had occurred and the cause of death was cardiac.

The external rupture was more difficult to find than the internal rupture, because of the great amount of blood infiltration of the surrounding tissues; in a number of cases no definite rupture was found at all in that site, a general blood infiltration of the periaortic coat having occurred, from which blood had gradually oozed into the surrounding tissues, particularly the mediastinum. In the strict sense, the "secondary" tear is a tearing of the outer part of the dissected media, exposing the adventitia, which is then infiltrated and often stripped off the underlying media for some distance (for example, Case XIX). Because of the weakness of the adventitia, this as a rule gives way eventually, most commonly into the pericardial sac, although the blood often reaches the latter by infiltrating the fatty tissue at the base of the heart and then rupturing through the visceral pericardial covering (for example, Cases VI and XXIV). Often the adventitia of the pulmonary artery is infiltrated.

The Region into which the Blood is Extravasated.

The region into which the blood is extravasated is of more importance than the manner in which it arrives there. In all the recent cases external rupture had occurred, except in Case XV, in which the patient died of heart failure. In eight cases rupture had occurred into the pericardial sac, which contained amounts of blood and blood clot varying from 12 to 40 ounces (12, 12, 12, 14, 15, 20, 26 and 40) with an average of 19 ounces; that is, in eight cases out of 18 external rupture had occurred into the pericardium. In Holland and Bayley's 19 cases[5] death was due in each case to rupture of the dissection; hæmopericardium occurred in seven cases. In Flaxman's 19 cases[2] death was caused by rupture of the aneurysm in 14 cases, and of these, in five a hæmopericardium was present. Shennan[1] reports in detail 13 out of 16 of his own cases, which he had met with at Aberdeen and Edinburgh; of these patients, nine died of hæmopericardium, three of a left hæmothorax, and one of acute congestive cardiac failure, due to the dissection. Possibly the three remaining cases (not included in his report) were of the old "silent" type, not causing death; anyhow, the number causing death by hæmopericardium was at least nine (and possibly a few more). Of Shennan's 300 cases collected from the literature, in 218 the dissections were recent, causing death. Of these, the dissection involved the intrapericardial portion of the aorta in 177 cases; among these, hæmopericardium occurred in 152 cases. External rupture elsewhere occurred in all of the remaining cases, except four, in which the cause of death was heart failure. Of the 41 recent dissections not involving the intrapericardial part of the aorta, 40 ruptured externally, not into the pericardium. Of Shennan's 79 cases of chronic or old dissections, in 16 the aneurysm ruptured into one of the body cavities, causing death (but insufficient details are given), and in the remainder death resulted from causes other than the dissection. Hence the number of deaths due to hæmopericardium was 152 (perhaps 168) or 66% (possibly 73%) of the 234 dissections causing death.

Thus, in from about 35% to 65% of cases the immediate cause of death due to the dissection was hæmopericardium.

Shennan also found that of the 177 intrapericardial dissections the 86 which affected only the ascending aorta, with or without a little of the first part of the arch, all led to hæmopericardium. Of six dissections of similar extent, at the Royal Adelaide Hospital, five led to hæmopericardium, and the sixth (Case IX), an old dissection, ruptured into the mediastinum.

In three of the 17 cases in which external rupture was recorded, the rupture was into the left pleural cavity—in two directly (Cases XI and XXI) through the parietal pleura from the arch or first part of the descending aorta, without infiltration of the mediastinum, and in one (Case XXIII) indirectly, first by gross infiltration of the mediastinum and then rupture through the pleura, and also infiltration behind the parietal portion of the pleura. In one case (Case VII) 30 ounces of blood had collected behind the left parietal pleura after much infiltration of the mediastinum, and without rupturing the pleura. In four of Holland and Bayley's[5] cases the rupture led to left hæmothorax. Thus, in four of 17 to 19 cases of dissecting aneurysm causing death, a left hæmothorax or blood behind the left pleura was present; according to Shennan,[1] in his analysis of his 300 cases, the figure is actually much lower than this.

The mediastinum was infiltrated in 12 cases of the 17 in which the aneurysm caused death, in three cases (Cases VII, IX, XXIII) grossly, and to a lesser extent in the others. From the mediastinum the hila of the lungs and base of the heart had become infiltrated in several cases, the neck tissues in one (Case IV) and the trachea and bronchi in one (Case XXII). Infiltration of the pulmonary artery occurred in four cases; in all of these the mediastinum was considerably infiltrated, and in three of them hæmopericardium was present. The infiltration of the periaortic coat and mediastinum must have given rise to much of the pain and shock associated with these dissections. In six of the twelve cases showing mediastinal infiltration and one showing much periaortic infiltration (Case XIII) the blood pressure after onset was very low or did not register; it was normal or only slightly low (100 millimetres of mercury, systolic, and 70, diastolic) in two cases; was high (180 millimetres of mercury, systolic, and 130, diastolic) in one case; and was not estimated in four cases. In six of the seven recent and recent-on-old cases in which the blood pressure after onset was low, infiltration of the periaortic coat or of the mediastinum or of both was present.

In one case (Case VIII) the external rupture was into the retroperitoneal tissues, including the kidneys.

Other sites of rupture have been described, but are rare. Holland and Bayley[5] describe a case (Case XVIII)

of several days' duration, in a male aged forty-six years, in which the entire length of the aorta was dissected, also the right common and external iliac arteries and part of the femoral artery, with secondary rupture, first into the thigh three inches below the inguinal ligament and then into the pericardium. This occurred shortly after a lumbar sympathetic nerve block was performed for a suspected thrombosis of the femoral artery. This case was also of interest in that the primary tear, 2·5 centimetres above the aortic valve, completely encircled the lumen, as in Case XXIV of this series.

Cardiac Hypertrophy and Other Observations.

The heart was hypertrophied in many instances. In thirteen cases in which the weight of the heart was recorded, it varied from twelve to twenty-two ounces, being sixteen ounces or over in eight cases; in six of the remaining cases "some" or "moderate" hypertrophy of the heart or left ventricle was described, "much" hypertrophy was described in one case, and in four no mention was made of the size of the heart. Hence in at least 15 out of 24 cases the heart was at least moderately hypertrophied. The hypertrophy was mostly of the concentric type in the left ventricle, due probably to high blood pressure. Of Flaxman's 19 cases[2] the heart in 16 weighed 16 ounces or more, and in nine of these 16 the weight was 20 ounces or more. The heart was also dilated in many of the cases in this series and in others the great veins were congested, the heart being prevented from dilating to any great extent by blood in the pericardium.

The liver, apart from being congested in most cases, had a "nutmeg" appearance in two.

The decapsulated kidney showed a fine granulation in six of the 24 cases; this is helpful evidence of possible high blood pressure during life.

A consideration of atheroma of the vessels generally does not reveal much, as, although there was often a great deal of atheroma of the abdominal aorta and of the vessels at the base of the brain, there was usually very little or none at the site of any primary tear.

In two cases the thyreoid was small and atrophied.

The post-mortem diagnosis of the causes of death has already been discussed; but it is of interest to recall that death has been due to a cardiac, vascular, or cerebrovascular cause in all cases except one, in which it was due to hypostatic pneumonia following cerebral softening.

Ætiology.

Predisposing Causes.

Only a few dissecting aneurysms can be definitely related to atheroma, and these are usually of a fairly localized type; those affecting the ascending aorta apparently have no relation to atheroma.

In some cases it is found at autopsy that the intima, which looks normal, is actually very soft and can be easily torn; such an intimal degeneration could explain a primary rupture of the intima and adjacent medial fibres, and this could be readily followed by dissection of the apparently normal media, as it is found that the latter can be relatively easily split longitudinally *post mortem*.

Holland and Bayley have given a good *résumé* of the various theories of the causation of dissecting aneurysm. They consider that the condition of *medianecrosis cystica* of the aorta, found microscopically in the often macroscopically normal aorta, is related to the longitudinal splitting of the media and the formation of a hæmatoma, and that the condition can be found in all cases of dissecting aneurysm in which "a diligent search is made". However, other investigators have found medianecrosis in many undissected and apparently normal aortas of people of the same age group. It is not intended to discuss here the microscopic findings of the aorta in this series of cases.

Arteriolar sclerosis of the media, causing fibrosis, has been put forward by some investigators as a cause of the lesion, but others have pointed out that the fibrosis which it causes, as in syphilitic aortitis, disfavours dissection by matting together the lamellæ; at least, syphilitic aortitis, if present, is merely coincidental.

Mesaortitis and medial degeneration have also been considered as the causative agents. Shennan thought that in some cases medial degeneration was due to a slow accumulation of "toxins", which also affected the intima.

The relation which long-continued high blood pressure has as a predisposing cause is not certain, though it probably plays a part, as evidenced by the records of its occurrence and of hypertrophied hearts in many cases.

The congenital predisposing causes are coarctation of the aorta and hypoplasia of the aorta. It has been stated that 20% of persons with the former condition die of spontaneous rupture of the aorta, with dissection; this fact favours long-continued high blood pressure as being a predisposing cause in some of the cases in which the aorta is macroscopically normal before dissection.

Immediate Causes.

Shennan has conveniently divided immediate causes into three groups: (*a*) external trauma, (*b*) sudden strain, (*c*) emotion. The two last-mentioned he groups together as internal trauma.

A history of recent severe trauma to the chest, abdomen or back is not common. In only one of these cases (Case IX) was such a history given, in this instance an injury to the chest; whereas the significance of such trauma is often difficult to assess, the relation in this case seems definite, especially when coupled with the post-mortem findings. A history of sudden emotion or physical strain is more difficult to obtain, especially the former, but is usually of more definite significance. A definite possibility of emotion was present in only one case (Case XIX). Shennan[1] suggests that the emotional strain of vivid dreams may be of importance.

A consideration of the physical occupation of the patients at the time of onset showed that fourteen cases occurred during the patient's waking hours, and seven during sleep or while he was in bed; in three cases no history could be obtained. In four cases of the first group the only information was that the onset occurred during waking; in six cases the onset occurred while the patient was walking (including one in which a man was looking in a shop window); while in three cases there was a definite strain, one patient straining at stool (Case XIV), one bending down (Case III) and one drying himself after a hot bath (Case VI); in the fourteenth case (Case XIX), already mentioned under emotion, there may also have been physical strain. Thus in nine cases out of 21 there was a definite history of at least moderate exertion. A history of a cough or chronic bronchitis was obtained in seven of the 18 recent and recent-upon-old cases, including six in which there was no history of other exertion. Flaxman[2] reported a case (Case IX) of a young man, aged twenty-two years, suffering from a respiratory infection, with a severe cough, who developed an extensive dissecting aneurysm; in this respect this case shows some resemblance to Case V of this series.

It has been found that a sudden large increase in pressure inside the aorta (and pulmonary artery) of a normal young person can lead to a complete rupture; and one such case, reported by J. B. Cleland,[6] in which both the aorta and pulmonary artery had completely ruptured through near their origins, causing almost immediate death, occurred in a young man as a result of a crush injury of the chest. Also, Shennan[1] reports the case (page 80) of "a small rupture through all the coats of the absolutely healthy aorta in the region of the *ductus arteriosus*, without any dissection, in a sailor who had climbed up on the glass roof of a railway station to retrieve his cap, and fallen through on to the platform". It is therefore conceivable that a sudden severe strain, in a person of middle age, without any particular pathological changes other than approaching senility, could also cause a partial rupture of the inner coats of the aorta, in its proximal part (where the pressure is highest) and dissection could easily follow. In some cases this had obviously occurred, but in others it is not at all certain that the so-called "primary" tear had actually been the first lesion and that it had not developed after the appearance of an aneurysmal bulge in the media.

In the cases in which no primary tear is found it has been suggested that a sudden strain may cause rupture of a *vasum vasorum* of the media, with formation of a spreading hæmatoma.

Clinical Symptoms and Signs.

In all of the 18 recent and recent-on-old cases the onset was quite sudden, which is probably the most characteristic feature of the symptomatology. Of these 18 cases, in 13 the presenting symptom was severe pain, in some cases excruciating, being in the chest alone in five cases, radiating to or from the chest in five cases (including radiation to the left arm in one instance), in the right leg in one case, in the lower part of the abdomen in one case, and the position was not described in one case; in four cases there was an absence of pain; but in two of these, the patients (Cases X and XX) suffered greatly from dyspnœa and orthopnœa, one (Case X) having also nausea and later loss of consciousness; a third suffered from nausea and dizziness (Case XXII) and a fourth became unconscious without any symptoms at all (Case XVII); in one case insufficient history could be obtained.

In one of the recent cases (Case VII) there was a history of three previous similar attacks within two months, but there was no mention at autopsy of any old dissection. As the heart muscle was pale (scarred?) these attacks might have been anginal attacks (the coronary arteries were not mentioned). In the one definite old active case (Case IX) the presence or amount of pain with the blow on the chest was not recorded.

Of the six old "silent" cases, in one, Case XIV, there was a history of violent pain (the situation is not stated) followed by loss of consciousness, while the patient was straining at stool, seven months before death. No history suggesting the time of onset was obtained in the other five cases.

Altogether in the recent cases consciousness was lost in three cases, two of the patients regaining consciousness, while three patients became semi-conscious after the onset.

Loss of power in the right arm occurred in one case (Case X), in which there was no pain; and numbness occurred in the left leg in another case (Case XV), abdominal pain being present.

Blood Pressure.—On examination after the onset of the attack three patients showed a low blood pressure (60 and 35, 90 and 45, and 90 and 35 millimetres of mercury, systolic and diastolic respectively), and in four patients the blood pressure was too low to register (in none of these latter cases was estimation of the blood pressure attempted on both arms, as it should have been). In two of these seven cases there was a history of high blood pressure before the attack. One patient had a slightly low blood pressure (100 and 70 millimetres of mercury, systolic and diastolic) and one a normal blood pressure; six had a high blood pressure, two having a previous history of high blood pressure; and in three cases the blood pressure was not taken. Also, of the eight patients with high blood pressure at or before examination, two were in their fifties, four in their sixties and two were over seventy years of age.

In the one old case (Case XIV) in which a history was obtained, the systolic blood pressure just after the attack was 170 millimetres of mercury and the diastolic 150 millimetres; just before death, seven months later, the blood pressure had fallen to 110 millimetres of mercury, systolic, and 80, diastolic, probably owing to a failing heart. The blood pressure was high at death in two old cases; no record of the blood pressure was noted in the other cases.

The Pulse.—The pulse during the attack in one of the recent cases was imperceptible; it was almost imperceptible in two cases, feeble in four cases, of good volume in one case, and the volume was not recorded in ten cases. The rate varied from 48 per minute to 140 per minute, with an average of 91 per minute.

Heart Sounds.—In the eight cases of hæmopericardium the heart sounds could not be heard in three cases, they were "very distant" in one case, they were "feeble" or "distant" in two cases, and were evidently easily heard in two cases (but in the last mentioned it is quite possible that the hæmopericardium had not formed at the time of examination). In another case, in which the mediastinum had become infiltrated (Case XVIII), the heart sounds were not heard; but, except for this case, the distant or absent heart sounds were due in all cases to hæmopericardium; in the remaining nine cases the heart sounds were easily heard in six, feebly in one, and there was no record in two cases.

Diagnosis.

The diagnosis of the recent attacks was fairly variable, and in none did it suggest the correct pathological cause of the disease, that is, in none was the dissection suspected before death. In six cases a diagnosis of coronary occlusion was made; in one case there was a diagnosis of coronary occlusion or ruptured peptic ulcer, or gastritis; in three cases the diagnosis was congestive or acute congestive cardiac failure; in one case the diagnosis was uræmia; in one case mediastinal tumour, in one case influenza; in one, bronchopneumonia; in two, cerebral thrombosis; in one, pulmonary hæmorrhage due to metastatic deposits; and in one, hæmatemesis. It is perhaps strange that, in a condition which can so resemble coronary occlusion in symptoms, death was not caused by it in any of the six old cases, although in three death was due to cerebral hæmorrhage and in one to cerebral softening; in none of the new or old cases was there at autopsy any sign of recent or old cardiac infarction.

In the diagnosis of the old cases an aortic aneurysm was suspected in Case I.

Treatment.

No special treatment was given, apart from rest in bed, morphine and trinitrin, except in one case (Case IX). In this instance the patient was sent to receive deep X-ray therapy, but died while waiting at the radiology department.

Period of Survival.

In the recent cases the length of history before death varied from three hours to eleven days, with an average of three days and three hours (seventy-five hours). Of the five patients who died in the first twenty-four hours, three had a hæmopericardium, one had a left hæmothorax, and one had much infiltration of the mediastinum, with a very low blood pressure. One patient with hæmopericardium died on the second day, two died on the third day, one on the fifth, and one on the eighth. Table II is a comparison of the length of history of the recent cases (*a*) in which death followed hæmopericardium or blood in or behind the left pleura, or much mediastinal infiltration, and (*b*) those in which none of these lesions was present.

TABLE II.

Day.	Number of Deaths Following Hæmopericardium *et cetera*.	Number of Deaths in which Hæmopericardium *et cetera* were Not Present.
1	5	—
2	3	—
3	1	1
4	1	—
5	1	1
6	—	1
7	1	1
8	—	—
9	1	—
10	—	—
11	—	—
12	1	—
Total ..	14	4

In the 18 recent and recent-on-old cases 12 patients died gradually, five died suddenly, and the mode of death was not recorded in one case.

Conclusion.

As these cases of dissecting aneurysm are not as rare as many people think they are (20 cases in the last 2,600 autopsies or 1 in 130; the incidence of recent cardiac infarction apart from other coronary accidents at autopsy, 82 cases or 1 in 32), it is as well to keep their possibility in mind, especially in the differential diagnosis of a coronary accident, ruptured gastric ulcer or other acute abdominal emergency. However, if all the thoracic dissecting aneurysms were diagnosed as coronary occlusion, the patients would, far from being the worse, probably benefit, as the treatment is the same in each case—absolute rest, morphine *et cetera*. It should also be remembered that, far from having a high blood pressure on admission, many patients have very low blood pressures.

The one clinical fact most likely to make a post-mortem examination desirable is sudden death, which is probably more frequent in dissecting aneurysms than in coronary occlusion, and it is probable that some patients diagnosed as suffering from coronary occlusion, and dying relatively slowly, are suffering from dissecting aneurysm.

TABLE III.

Grouping of the Twenty-four Cases of Dissecting Aneurysm.

Type and Site.	Number of Cases.
Old smooth-lined double channel present. Case I ..	1
More recent dissections:	
Ascending aorta to at least cœliac axis. Cases III, IV, VII, XV, XVIII, XIX, XXI	7
Ascending aorta chiefly. Cases IX, X, XIII, XVI, XX, XXII, XXIV	7
Ascending aorta to commencement of descending. Case XVII	1
Arch to bifurcation. Case XXIII	1
Arch to descending aorta. Cases XI, XII	2
Descending thoracic aorta. Case XIV	1
Abdominal aorta just above bifurcation. Case V ..	1
Abdominal aorta. Cases VI, VIII (ruptured) ..	2
Right common iliac, healed. Case II	1
Total	24

Acknowledgement.

I am very grateful to Professor J. B. Cleland for advice and help and for placing at my disposal the tabulated results of the pathological lesions met with at autopsies at the Royal Adelaide Hospital, as published in *The Medical and Scientific Archives of the (Royal) Adelaide Hospital*. This source of information led to easy access to the clinical notes and to the full post-mortem record. I am indebted also to the various honorary physicians and surgeons under whom the patients were, and to those responsible for the post-mortem examinations, particularly Professor Cleland and Dr. J. B. Thiersch.

Selected Reports of Cases.

CASE I.—This case was reported on more fully by J. B. Cleland in THE MEDICAL JOURNAL OF AUSTRALIA of April 9, 1927, at page 538, and is also included in Shennan's 300 cases of dissecting aneurysm collected from the literature up to 1933.[a)]

A male patient, aged fifty-five years, was admitted to hospital on August 14, 1925, under the care of Dr. F. S. Hone. He had been bedridden for the past five years, and was believed to have an aortic aneurysm. On examination of the patient, the apex beat was situated in the seventh intercostal space at the anterior axillary line, and was of a heaving nature; a systolic thrill could be felt over the right side of the chest, and a systolic murmur could be heard all over the chest. The patient developed a bedsore, became moribund and died eleven days after his admission to hospital.

Autopsy Number 169/25 (J. B. Cleland).—A dissecting aneurysm was found extending from the aortic cusps, along the anterior wall of the ascending aorta, and along the posterior wall of the descending aorta, certainly down to the diaphragm, and possibly to the bifurcation. (The condition was discovered only in the retained specimen of heart and aorta, which was cut off at the diaphragm.) The dilated upper end of the dissecting aneurysm communicated with the true lumen of the aorta through a small hole just above the anterior aortic cusp, and the lower end probably also had communicated with the lumen, for the walls of the new channel were smoothly lined with endothelium as in the true lumen, and blood had obviously been circulating in the false channel for some time. There was only slight atheroma of the aorta. The heart was hypertrophied and dilated, and the cut surface of the liver presented a somewhat nutmeg appearance. Some straw-coloured fluid was present in both pleural cavities.

CASE III.—A large, well-nourished female patient, aged sixty-one years, was admitted to hospital on July 12, 1932, under the care of Dr. A. R. Southwood. She had felt unwell for the last three years, being tired and breathless on moderate exertion. She had been taking thyreoid tablets for some time, and two weeks previously she had started to take extra tablets, because she began to feel worse. Twenty-four hours before her admission to hospital, while bending down, she felt a sudden excruciating pain, starting in the centre of her abdomen, and passing up her chest and neck and down her left arm; this pain lasted about one hour, and was followed after an injection of morphine by a dull epigastric pain.

On examination, the patient was dyspnœic and her face was cold and of an earthy, cyanosed colour. The pulse was feeble, the rate being 48 per minute. The apex beat was not palpable, but the area of cardiac dulness extended transversely from one inch to the right of the right sternal margin to eight and a half inches to the left of the mid-sternal line in the second intercostal space; the heart sounds were distant and regular, but could be heard only at the base of the heart. The blood pressure could not be ascertained. The percussion note was slightly impaired all over the posterior part of the right side of the chest. The urine contained much albumin.

A diagnosis of acute congestive cardiac failure was made. The patient remained in a distressed condition and died suddenly while being washed three days after the onset (two days after her admission to hospital).

Autopsy Number 105/32 (J. B. Cleland).—A dissecting aneurysm, extending throughout the whole of the aorta and common iliac arteries, formed a new channel in the layers of the anterior wall, with considerable dilatation of the aneurysm at the level at which the ascending aorta crossed in front of the pulmonary artery; here the dilated portion had ruptured into the pericardial sac, which contained twelve ounces of blood clot and several ounces of serum. In the intima and inner part of the media of the anterior wall of the ascending aorta, four centimetres above the aortic valve, was an almost rectangular tear, measuring two centimetres by one centimetre, communicating with the aneurysm. Blood was extravasated into the mediastinal tissues and along a second new channel in front of the descending aorta. Considerable atheroma of the thoracic aorta was present (the amount at the site of the tear was not stated), and much atheroma was found in the abdominal aorta and common iliac arteries. The heart muscle was pale, and showed a great deal of fatty infiltration and fatty degeneration, being almost replaced in parts by fat; the size of the heart was not stated. The renal cortex was unusually pale, and the thyreoid was atrophied. The lungs were compressed in their lower parts and emphysematous in their upper parts; blood had infiltrated the hila. The liver was deeply congested.

CASE IX.—A male patient, aged thirty-five years, was admitted to hospital on April 26, 1939, under the care of Dr. K. S. Hetzel. For about two weeks his face and neck had been "puffy", especially in the mornings. Four days before his admission to hospital he had felt a sharp, sudden pain in his right leg, and noticed that almost immediately afterwards his scrotum became swollen. Then, two days before his admission to hospital, his thighs had become swollen, and the next day his right arm also became swollen. About three to four months earlier he had received a severe blow on the right side of his chest from an 84-pound tin of paint, and he had had a cough ever since when he lay on his back.

On examination, the patient was a sallow-complexioned, youngish man, sitting up, and in some respiratory distress; the eyelids were rather puffy. The pulse rate was 140 per minute, and the blood pressure was 100 millimetres of mercury (systolic) and 70 millimetres (diastolic). In the chest, both sides of which moved evenly with respiration, the percussion note was impaired on the right side "posteriorly and anteriorly". The breath sounds were absent

over the base of the right lung posteriorly, and were diminished in the right axilla, where they were of a cogwheel type; the vocal fremitus was normal. There was no mention of the heart in the examination. A lumbar pad was present with œdema of both arms and forearms, of the scrotum and penis, and of the lower parts of both thighs, but not of the legs or feet.

A clinical diagnosis of possible mediastinal tumour was made. On the day after the patient's admission to hospital, his pulse rate was 90 per minute, but the heart rate by auscultation was 150 to 160 per minute. On the second day after his admission to hospital he was sent down to the radium clinic for deep X-ray therapy, but died suddenly five minutes after his arrival there.

Autopsy Number 77/39 (J. B. Thiersch and T. J. Constance).—A large hæmorrhagic mass was found in the anterior part of the mediastinum, when the chest was opened. There was a large dissecting aneurysm, about four inches (ten centimetres) long, of the ascending aorta, commencing about one inch (2·5 centimetres) above the aortic valve; a small communication (the age and site of which were not recorded) three millimetres in diameter was found between the aneurysm and the original lumen of the aorta; "hard calcification" was present in the wall of the aorta and aneurysm; the blood in the anterior part of the mediastinum was partly fibrosed and partly fresh (twenty ounces). The heart was not dilated (it contained four ounces of blood), but the size was not recorded; the heart muscle was brown, and some calcification of the coronary arteries was noted; the pericardium was universally adherent. There was no mention of atheroma of the aorta. The superior *vena cava* had become compressed, and was nearly perforated, by the aneurysm. The liver, spleen, kidneys and pancreas were congested, and the liver was affected by a fine cirrhosis.

CASE XI.—A female patient, aged sixty-nine years, was admitted to hospital on August 11, 1939, under the care of Dr. S. R. Burston. One week before her admission to hospital she had had a severe attack of "influenza" from which she had recovered; but she had "relapsed" again on the day before her admission to hospital. When admitted to hospital she felt weak and had pain in the front and sides of her chest, and she had been suffering from nausea and retching since her "relapse". She had been tired and breathless on exertion for some time.

On examination, the patient was dyspnœic and cyanosed, but the facies was not "toxic". The pulse rate was 105 per minute and the blood pressure was 210 millimetres of mercury (systolic) and 120 millimetres (diastolic). The chest moved evenly on respiration. The apex beat was felt in the fifth intercostal space in the mid-clavicular line; the percussion note was not impaired in the second left intercostal space; no bruits were detected, but the second aortic sound was particularly loud. There was slight impairment of the percussion note and of the breath sounds at the base of the left lung posteriorly, with some crepitations.

On the fourth day after her admission to hospital the patient had no pain, but she still had a slight cough; the percussion note was impaired at the base of the left lung posteriorly, where the breath sounds were almost absent, and was diminished in the left axilla. The blood pressure was 135 millimetres of mercury (systolic) and 85 millimetres (diastolic). The temperature was 100° C., and a course of "M & B 693" treatment was commenced. At 5.40 a.m. on the seventh day after her admission to hospital she complained of a sudden severe pain in the centre of her chest; she became breathless and almost pulseless. The volume of the pulse soon returned a little, but she remained cyanosed and shocked, and her pulse and respiration rates were increasing. On examination of the patient, the percussion note was much impaired all over the chest. She died at 5.45 p.m. on the same day (August 18), twelve hours after the onset of the sudden attack, the pulse rate having risen to 150 per minute and the respiration rate to 50 per minute.

Autopsy Number 190/39 (R. H. Binns).—A large atheromatous ulcer, 2·5 centimetres in diameter, was found at the junction of the aortic arch and the descending aorta; the bed of this ulcer had ruptured and caused dissection of the coats of the aorta for several inches around. A large hæmatoma had been formed in the periaortic tissue near the hilum of the left lung, and had burst into the left pleural cavity (which contained 35 ounces of blood), causing the left lung to collapse. There was slight atheroma in the ascending aorta, and large atheromatous patches were present in the aortic arch and in the descending aorta. The heart muscle and coronary arteries were healthy and the heart was "of normal size". The stripped surfaces of the kidneys, which were also of normal size, were finely granular. The lungs were normal, except for compression of the left lung.

CASE XV.—A male patient, aged twenty-seven years, was admitted to hospital at 12.50 p.m. on May 2, 1942, under the care of Dr. Guy Lendon. The diagnosis of bronchopneumonia was made. Thirty-six hours before his admission to hospital he had experienced a "tight" feeling around his chest, accompanied by breathlessness and by numbness in his left leg, and since then he had had pains in his "stomach". He had vomited two or three times, and had had a cough, bringing up some blood-stained sputum.

On examination, the patient was a sick-looking young man, with cyanosed lips and in some respiratory distress. His pulse was feeble, the rate being 152 per minute; the respirations numbered 40 per minute. The apex beat was felt in the fifth intercostal space within the nipple line; the heart sounds were loud, rapid and regular. The breath sounds were harsh, and râles and crepitations were heard scattered over both lung fields.

On the first day after his admission to hospital he was still very ill and cyanosed, and had a cough; his blood pressure was 90 millimetres of mercury (systolic) and 45 millimetres (diastolic). On the second day after his admission to hospital a systolic murmur was heard at the apex; the blood pressure was 105 millimetres of mercury (systolic) and 50 millimetres (diastolic), the pulse rate was 124 per minute, and the respirations numbered 28 per minute; digitalis treatment was commenced. On the third day after his admission to hospital a systolic thrill could be felt at the apex, and the apical systolic murmur was heard to be louder. The liver edge was felt to be three inches (7·5 centimetres) below the right costal margin. It was thought that his condition might possibly be a state of suprarenal cortical collapse, and he was put on a diet rich in salt. On the fourth day after his admission to hospital his condition had much deteriorated; his temperature became subnormal, and his pulse rate was 108 per minute; he was given cortical extract. He died six and a half days after the onset of the illness (five days after his admission to hospital).

Autopsy Number 131/42 (J. B. Cleland).—A dissecting aneurysm was found extending from the aortic valve to the end of the common iliac arteries, with valve-like openings into the commencement of the innominate, right common carotid, left subclavian, and both internal iliac arteries; in the aortic arch the anterior or inferior wall was not dissected: the state of the blood clot in the dissected coats was not recorded. There was no obvious aperture in the intima. The heart was hypertrophied (it weighed 18·5 ounces) and dilated, and ante-mortem clot was present in the left auricular appendix; coronary atheroma was present, but the heart muscle was of good colour and firm texture. There was no mention of atheroma of the aorta. The lungs did not collapse at all when the chest was opened, and were œdematous and congested, being friable at the bases; no ante-mortem clots were found in the pulmonary artery. The cut surface of the liver presented a somewhat nutmeg appearance. There was an infarct in the right kidney. Some ante-mortem clots were found in the pampiniform plexus of veins, and the left testis was very congested. There was plenty of lipoid in the suprarenals.

CASE XVII.—An elderly looking man, aged sixty-one years, was admitted to hospital at 3 p.m. on July 24, 1942, under the care of Dr. H. K. Fry. While looking in a shop window he had suddenly lost consciousness; before this he had had no pain or queer sensation. He was immediately taken to hospital, and on his arrival could be roused to answer questions, but was still without pain. He had been told by his doctor a week previously that he had very high blood pressure.

On examination of the patient the breathing was stertorous and noisy, the respirations numbered twenty per minute and the skin was cold and moist; the pulse rate was 82 per minute and the blood pressure was 90 millimetres of mercury (systolic) and 35 millimetres (diastolic). The pupils were equal and reacted normally to light. The apex beat was felt in the sixth intercostal space, four and a half inches (eleven centimetres) from the mid-sternal line; the second heart sound was absent once in about every eighth beat. No abnormality was detected in the lungs. The limbs were flaccid, and the tendon jerks could not be elicited; but the plantar responses were flexor in type. The clinical diagnosis of a coronary occlusion was made. The patient died eight hours after the onset of the illness (and his admission to hospital).

Autopsy Number 241/42 (J. B. Cleland).—A transverse tear (it was not stated whether it was sharp or ragged) about eight centimetres long was found in the posterior wall of the ascending aorta, three centimetres above the aortic valve, and involving the intima and the inner part of the media. The outer part of the media was separated from the inner part by fresh blood clots from the aortic valve down to the first few inches of the descending aorta. Extravasation of blood had occurred into the mediastinum and into the adventitia round the roots of the aorta and pulmonary artery within the pericardial sac. The heart was hypertrophied and dilated and considerable atheroma and some calcification of the coronary arteries were noted. There were some slight patches of atheroma above and around the origin of the right coronary artery, but none at the site of rupture. A little atheroma of the abdominal aorta was present. The liver and kidneys were congested.

CASE XXI.—A middle-aged-looking man of fifty-eight years was admitted to a surgical ward at 1.30 a.m. on January 2, 1944, under the care of Dr. L. C. E. Lindon. At 11 p.m., two and a half hours previously, he had experienced a sudden severe pain in his chest; it shifted down to his abdomen and caused him to groan a lot, and he had vomited a small amount of clear fluid, not blood-stained. He had had no recent dyspeptic symptoms, but he had had a ruptured peptic ulcer in 1936, followed by several severe hæmatemeses in the next few years. His blood pressure in 1942 was 185 millimetres of mercury (systolic) and 120 millimetres (diastolic).

On examination of the patient he had not much dyspnœa, but he was in considerable pain and his skin was cold and moist. The pulse rate was 84 per minute and the blood pressure was 210 millimetres of mercury (systolic) and 100 millimetres (diastolic). The apex beat was not mentioned. The heart sounds were clear and regular and the lung fields were "clinically clear". Some tenderness was present in the epigastrium, but no muscle guarding. The urine, of specific gravity 1,010, contained a trace of albumin.

The clinical diagnosis lay between a ruptured peptic ulcer, acute gastritis and a coronary occlusion. On the second day after his admission to hospital he had a recurrence of the chest pain, which was eased with morphine (as was the first pain). On the third day after his admission to hospital he began to have abdominal distension and to belch wind. On the fourth day after his admission to hospital he passed some blood clots in his fæces, and he still had occasional chest pains, which continued on and off until his death at 5.45 a.m. on the eleventh day after the onset of the disease. His pulse and respiration rates remained 80 to 90 per minute and 20 per minute respectively throughout his illness.

Autopsy Number 9/44 (J. B. Thiersch).—A dissecting aneurysm was found extending throughout the whole of the aorta: there was no tear in the intima, but the aneurysm appeared to have commenced at the arch of the aorta where it had formed a bulging cavity in the wall near the left lung, and had ruptured into the left pleura, which was filled with blood clot, compressing the left lung. Some atheroma (the situation of which was not stated) of the aorta was present with slight syphilitic aortitis. Much hypertrophy and dilatation of the heart were observed (it weighed 21 ounces) with some patchy atheroma of the coronary arteries and some scarring of the heart muscle. Some compensatory emphysema of the right lung was present. The right side of the diaphragm was at the level of the seventh intercostal space and the left at the level of the sixth intercostal space in the mid-clavicular line. Arteriolarsclerosis was present in the kidneys (which were rather small, red and granular), in the spleen and in the liver. There was a large butterfly ulcer of the duodenum and a gastro-jejunostomy was present.

CASE XXIII.—A pale, well-nourished man, aged seventy-six years, was admitted to a surgical ward at 5.15 p.m. on June 18, 1944, under the care of Dr. I. B. Jose. Five days previously, while chopping wood, he had lost some blood from his bowel; on the morning of the day of his admission to hospital he had had a sudden sharp pain in the left side of his chest and had felt very ill; he thought that he had passed more blood from his bowel. (The records do not state what he was doing at the time of onset.) He had recently had a cough with a good deal of sputum.

On examination the patient was pale; he was lying in bed, semicomatose and dyspnœic. The pulse at the wrist was almost impalpable, the rate being 128 per minute, and the blood pressure could not be registered. The percussion note was impaired at the base of the left lung and the breath sounds were not heard over the left lung area; this suggested the possibility of fluid in the pleural cavity. On rectal examination the prostate felt large and craggy. The clinical diagnosis was considered to be carcinoma of the prostate, with intrathoracic hæmorrhage, possibly due to metastatic deposits in the lung from the prostate. He was given saline solution and 500 millilitres of blood intravenously. A needle passed into the left pleura withdrew blood-stained fluid. He became restless during the night and coughed a little; he complained of being cold and slept very little. His pulse rate remained at about 120 per minute, and his respirations numbered 24 to 28 per minute. He gradually sank, and died at 5.45 a.m., twelve hours after his admission to hospital (eighteen to twenty-four hours after the onset of the attack).

Autopsy Number 153/44 (J. B. Cleland and I. A. Hamilton).—A rather triangular-shaped irregular tear was found in the intima and the inner part of the media of the terminal part of the aortic arch, with separation of the outer third of the media from the inner two-thirds by fresh blood clot; the tear extended down to the right common iliac artery on the right side, and not quite so far on the left side. From outside the aorta an aneurysmal bulge could be seen in the periaortic tissue on the left side of the arch; this was due to infiltration of the periaortic tissue with a layer of fresh blood clot 2·5 centimetres thick; from this bulge blood had infiltrated into the mediastinum (the anterior part of which was swollen with blood) and thence into the left pleural cavity, which contained 68 ounces of blood and blood clot; the left lung was compressed and the diaphragm on the left side was caused to bulge downwards. There was also separation by blood clot of the left posterior parietal pleura from the parietes opposite the axilla. The dissection of the media did not extend down so far as the aortic valve, but the infiltration of the periaortic tissues extended down to the subpericardial tissue covering the base of the heart. A slight amount of blood-stained fluid was present in the pericardial sac. The heart was of normal size (it weighed twelve ounces), the heart muscle was pale but firm, and some atheroma of the left coronary artery with a little rigidity was detected. There was no mention of atheroma of the aorta. A large carcinoma of the prostate was present, but no sign of secondary deposits was detected in the liver or anywhere else; the rectum was normal. The sternal marrow was pale.

CASE XXIV.—A well-nourished woman, aged fifty-three years, was admitted to hospital at 9 p.m. on July 31, 1944, under the care of Dr. Guy Lendon. Her daughter said that she had been well until just before her admission to hospital, when she found her on her knees in the passage, moaning and semicomatose, with staring eyes.

On examination, the patient was stuporose and somewhat cyanosed, the limbs especially being cold and blue. The pulse rate was 98 per minute and the pulse was of poor volume; the blood pressure would not register. The apex beat was impalpable, and the heart sounds could not be heard. No evidence of disease was found in the lungs. The pupillary reflexes were normal, as were the upper limb reflexes; the right ankle jerk could not be obtained, and the plantar responses were equivocal. The left arm did not move at all. On the day after her admission to hospital the patient recovered consciousness slightly, but she died suddenly at 1.15 p.m. (about forty-one hours after the onset of the attack).

Autopsy Number 193/44 (J. B. Cleland and W. F. Emery).—A clean-cut transverse tear of the intima and inner part of the media was found in the ascending aorta, 2·5 centimetres above the aortic valve, and completely encircling the aorta; the outer few laminæ of the media were separated from the majority of those of the inner part of the media by fresh blood clot, extending from the aortic valve up to the innominate artery. (There was no evidence of any dissection of the origin of the arteries arising from the arch.) Blood had leaked into the periaortic coat of the ascending aorta by way of a second tear of the outer part of the dissected media, and into the adventitia of the pulmonary artery and the wall of the right auricle, from which site blood had leaked through a small tear of the visceral pericardial covering into the pericardial sac, which contained fifteen ounces of blood and blood clot. Some blood had infiltrated the adjacent mediastinal tissues. Moderate hypertrophy of the left ventricle was present (the heart was not weighed); the coronary arteries were not examined, but the heart muscle appeared normal. Very little atheroma of the ascending aorta and the aortic arch was detected, and there was none in the region of the tear; but much atheroma was present in the abdominal aorta. Intense atheroma was present in the vessels at the base of the brain. The kidneys were normal.

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TYPING OF CORYNEBACTERIUM DIPHTHERIÆ IN QUEENSLAND.

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SINCE the discovery by Anderson *et alii* (1931 *et cetera*)[1] of the three types of *Corynebacterium diphtheriæ* and subsequent investigations of the type characteristics since that time, bacteriologists have carefully recorded the incidence of the various types in different parts of the world and attempted to correlate type with clinical severity. Some have found that the *gravis* type is the most severe (as originally suggested); but others have shown that types *mitis* and *intermedius* can be equally as virulent, so that it is now generally agreed that the original terminology of the types has little significance from the clinical point of view of severity. Latest American opinion[2] now tends to classify *Corynebacterium diphtheria* into the following two categories: (i) "*Corynebacterium diphtheriæ* type *gravis*", provided all the recognized *gravis* characteristics are exhibited; (ii) *Corynebacterium diphtheriæ* type indeterminate, but with the following *gravis* characteristics".

Study of the colony growth and morphology on the old Clauberg formulæ was difficult, because variation in quality and consistency of the media seriously interfered with colony formation, and it has been frequently noted that well-established standard *gravis* types, whilst growing characteristically on one Clauberg medium, will yield, when subcultured onto another batch of Clauberg medium, even a *mitis*-like growth. This difficulty has been practically eliminated by the use of the 10% guinea-pig blood medium formulated by Goldsworthy and Wilson,[3] and it is now a simple matter to classify *Corynebacterium diphtheriæ* types once the organisms have been isolated in pure culture.

A survey of available literature shows that most States of Australia have recorded at some time or other the incidence of the various *Corynebacterium diphtheriæ* types; but to date there is no similar record for the State of Queensland, and with this in mind it seems justifiable to publish the following investigations.

During the period from February to September, 1941, all swabs received at this laboratory and found to yield *Corynebacterium diphtheriæ* were thoroughly investigated to determine the types prevalent in southern Queensland. The swabs were inoculated onto tellurite blood agar medium (V. Glass),[4] and after twenty-four hours' incubation, colonies of *Corynebacterium diphtheriæ* were picked off and grown in pure culture on inspissated serum slopes. The pure cultures were then transferred to sugars (glucose, saccharose, starch), to nutrient broth, and to 10% guinea-pig blood agar (Goldsworthy and Wilson)[3] to determine whether they were of *gravis*, *mitis* or *intermedius* types.

A series of 51 swabs from patients suffering from diphtheria at the Brisbane General Hospital were included in this survey (per courtesy of Mr. N. R. Henry). Altogether 140 cultures came from patients suffering from clinical diphtheria and 60 from carriers, representing 198 persons. Tables I and II show the number of positive cultures obtained each month, the number and percentage of types amongst these, and the distribution throughout southern Queensland.

TABLE II.

Geographical Incidence of Types of Corynebacterium Diphtheriæ.

Locality.	*Gravis.*	*Intermedius.*	*Mitis.*	Total.
Brisbane General Hospital ..	27	—	24	51
Brisbane Area ..	24	—	20	44
Bundaberg ..	3	—	13	16
Maryborough	3	—	3	6
Gympie	7	—	5	12
Nambour	3	—	7	10
Kilcoy	—	—	3	3
Kingaroy	—	—	1	1
Esk	—	—	4	4
Ipswich	30	—	6	36
Boonah	1	—	1	2
Charleville	8	—	1	9
Southport Area ..	3	—	3	6
	109 (54·5% of all types)	Nil	91 (45·5% of all types)	200

It is interesting to note that no *Corynebacterium diphtheriæ* type *intermedius* was encountered (similar to that found in Adelaide by Puckey).[5]

When the series is considered as a whole, *gravis* predominated slightly over *mitis*, and this finding held also for the Brisbane area. At Ipswich there was a marked predominance of *gravis* and at Bundaberg of *mitis*. One *gravis* type (atypical) was met; its growth on guinea-pig blood agar was smooth and shiny (*mitis*-like), but all other characteristics were of *gravis* type. In two instances both *gravis* and *mitis* types were isolated, in one instance from a patient with clinical diphtheria and in one from a carrier.

TABLE I.

Monthly Incidence of Types of Corynebacterium Diphtheriæ.

Month.	Cultures from Patients.			Cultures from Carriers.			Total Cultures.
	Gravis.	*Intermedius.*	*Mitis.*	*Gravis.*	*Intermedius.*	*Mitis.*	
February	2	—	2	3	—	2	9
March	3	—	4	4	—	1	12
April	7	—	3	7	—	6	23
May	17	—	12	6	—	5	40
June	21	—	22	13	—	1	57
July	14	—	18	4	—	5	41
August	5	—	4	—	—	3	12
September	3	—	3	—	—	—	6
	72 (or 51% of patients)	Nil	68 (or 49% of patients)	37 (or 61% of carriers)	Nil	23 (or 39% of carriers)	200

ILLUSTRATIONS TO THE ARTICLE BY Dr. C. H. WICKHAM LAWES.

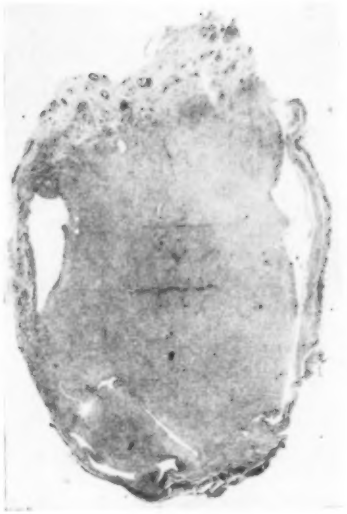

FIGURE IV.

Low-power photomicrograph. The whole cross section of the vein is seen. The greater part of the lumen is occluded, but recanalization is seen on the left hand side and at the lower pole.

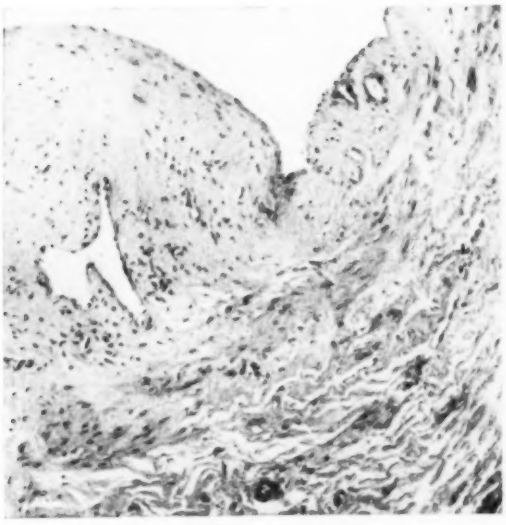

FIGURE V.

Photomicrograph (magnification × 100). The greater part of the section is occupied by fibrous tissue with cholesterol clefts. Portion of a large area of recanalization is seen at the top and a complete smaller area on the left. Note the endothelial lining of these channels.

FIGURE II.

FIGURE III.

FIGURE XIV.

ILLUSTRATIONS TO THE ARTICLE BY Dr. J. D. HICKS.

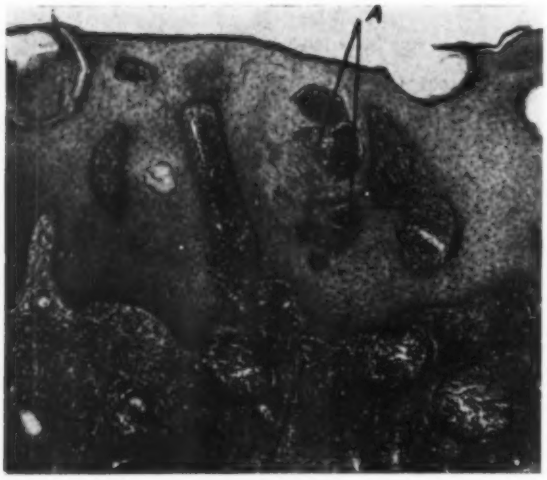

Figure I.

The irregularly thickened epithelium has a scaling surface. Intraepithelial abscesses are seen at A. The dermis is thickly infiltrated with cells, amongst which the pale pseudofollicles (B) stand out. Giant cells (C) are prominent. (× 100.)

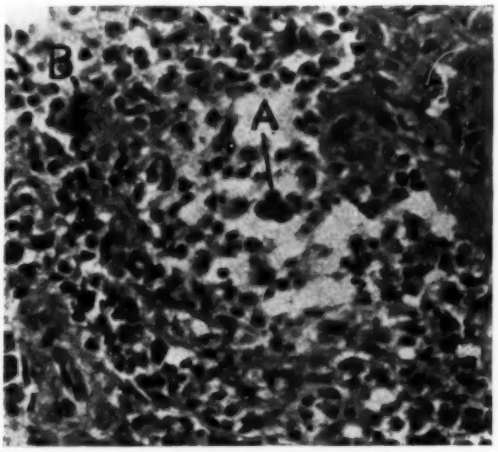

Figure II.

In the centre is a group of three of the typical organisms (A). Polymorphonuclear leucocytes, plasma cells and epithelioid cells loosely surround them. At B a giant cell is forming. (× 400.)

ILLUSTRATIONS TO THE ARTICLE BY Dr. EDGAR STEPHEN AND Dr. DOUGLAS REYE.

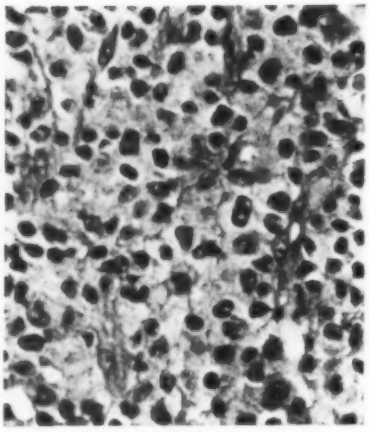

Figure I.

From the biopsy specimen. The cells are arranged in trabeculæ; they are fairly uniform in size and shape and many of them have a sharply defined outline. (× 200.)

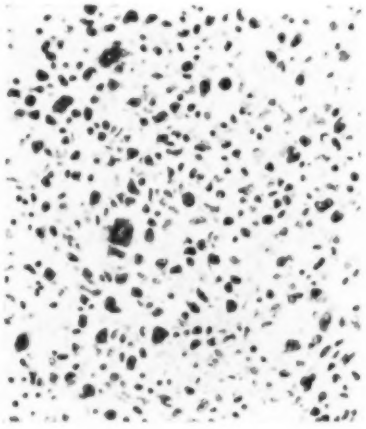

Figure II.

Portion of a mediastinal lymph node. The pleomorphism of the cells is apparent and forms a striking contrast to the cytological picture presented in Figure I. (× 200.)

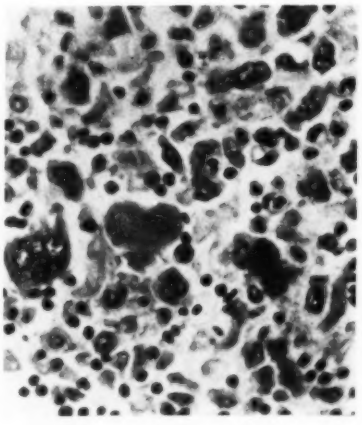

Figure III.

Another portion of the node shown in Figure II under higher magnification. The large hyperchromatic cells are clearly visible and some of these are multinucleated. The small, darkly staining points are lymphocytes and the occasional small cells with indented nuclei are eosinophile leucocytes. Two histiocytes, which appear pale in contrast to the other cells in the field, are to be seen towards the lower left corner. (× 400.)

Acknowledgements.

I wish to thank the Director-General of Health and Medical Services for Queensland, Dr. J. Coffey, for permission to publish this article; Mr. N. Henry, bacteriologist, for making available diphtheria cultures from Brisbane General Hospital patients; Dr. W. C. Sawers, of the School of Public Health and Tropical Medicine, Sydney, for kindly providing the standard type cultures *gravis*, *mitis* and *intermedius* for comparison; and last but not least, Dr. E. H. Derrick for constructive criticism and helpful guidance.

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(3) N. E. Goldsworthy and H. Wilson: "The Use of Blood Agar for the Identification of the Types of C. Diphtheriæ", *The Journal of Pathology and Bacteriology*, Volume XLVIII, January, 1939, page 125.

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(5) M. C. Puckey: "A Preliminary Survey of the Types of Corynebacterium Diphtheriæ Isolated at the Metropolitan Infectious Diseases Hospital, Northfield", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1938, page 851.

THE FATHER OF AMBROSE PRATT: THE PROBLEMS OF A PIONEERING COUNTRY DOCTOR.

By RICHARD T. KENNEDY,
Sydney.

A PRETTY little blonde, firm of mouth, stubborn of chin and aged but twenty-one years, Miss Caroline Kershaw advertised as follows in the *Gundagai Times* of September, 1870:

> MISS KERSHAW
>
> Begs to intimate that she has opened a Night School for young girls so that those who are unable to attend during the day can have the benefit of receiving instructions from 6.30 to 8.30 four nights in the week. Reading, writing, arithmetic, English grammar, geography, history, etc. Terms 15s. per quarter in advance.

The ambitious and diminutive schoolma'am soon found her nights otherwise engaged than in teaching the elements of education to the young ladies of Gundagai, for her spare evenings were quickly monopolized by the young, good-looking English doctor who had recently settled in that township. Tall, dark, handsome and clever, he had all the attributes that would appeal to the charming teacher.

He was Eustace Henry Lever Pratt, M.D., only son of H. F. A. Pratt, M.D., F.R.S., Count of the Holy Roman Empire and grandson of Sir John Hesketh Lethbridge, baronet, of Sandhill Park, Somerset. Dr. Henry Eustace Lever Pratt came to Gundagai at the age of twenty-nine years in 1870.

Dr. Pratt and Caroline Kershaw were married in the same year. The ceremony was performed first by the local priest and a few minutes later by the local parson. So, apparently, were their individual religious qualms satisfied.

The honeymoon, spent in Goulburn, was short and sweet, for the young doctor had great responsibilities as medical officer to the Gundagai Benevolent Society's hospital and could not be long away from his practice. The happy couple returned to Gundagai to the tune of wild tin-kettling—an unlawful practice that brought down the stern reprimand of the local newspaper.

A further reason for the doctor's anxiety to return was that the Gundagai Hospital was wallowing in the slough of financial despond. The doctor felt that it was his duty to remain at his post to help keep the hospital going. The year 1870 had shown that the hospital had attended the enormous number, for those days, of 45 patients in the twelve months. The expenses amounted to £428, to meet which the glum treasurer announced he held but £371. Things certainly looked gloomy, and there were cheerless prognostications that the hospital would have to close after twelve years of useful existence. The blame was laid at the door of public apathy; people just did not seem to bother to contribute to such a worthy institution. In fact, some members of the public spoke in very severe terms of the reason for their apathy. One man wrote to the newspaper as follows:

> In a late issue you came out strong about hospitals and hinted at the very early closing of the Gundagai Hospital. No wonder it may close when able and healthy men are allowed to sponge on its funds. By looking over the register I find that one stout man is an inmate who attends church every Sunday night and walks around the town and pays his friends a visit!

However, for all the conscientious striving of the young doctor to counteract the indolence shown by the Gundagai, Adelong and Tumut residents, whose only hospital was at Gundagai, the doors had to be closed on September 1, 1871. They remained closed for exactly one year. The people of the district were much incommoded by this fact; but having brought it upon themselves they had no alternative but to manage as best they could.

The ailing poor were badly hit. It became necessary to have them committed to and incarcerated in the Gundagai gaol, so that accommodation and medical attendance could be provided for those most severely in need. An extract from the *Gundagai Times* for October, 1871, will give some idea of the straits to which the poor were driven:

> On Friday afternoon an inquest was held on the body of Charles Mercer, who died at the Gundagai gaol on Thursday morning.
>
> The evidence of Mr. J. Benton, gaoler, disclosed that the deceased, who was brought into gaol on Wednesday for safe custody, seemed in great pain and screamed and moaned incessantly. Dr. Pratt was called in, but deceased expired.
>
> There were four other prisoners in the cell with the deceased. Benton stated that he was obliged to admit sick persons or even those who were incurable, as there was no other place where they could be placed. The gaol was almost an hospital, and persons who had loathsome ulcers or other offensive diseases were often placed with other inmates of the prison cells; and the gaoler and warder had to sit up by night as well as to do duty by day!

On March 16, 1872, "a destitute man named Michael Lawlor, who had fallen down a high bank near Coolac and so injured himself that he lay helpless and unable to move, was brought into town and lodged in the lock-up as the only available place, which place, as it contained 17 prisoners, was most unfit for the reception of an injured man".

In August the attention of the hospital committee was directed to another deplorable case of misery and destitution, and the quotation is again from the local journal.

> A family named Smith have for some time past been living in a damp, wretched hut near Spring Flat, and no less than seven of them are suffering from typhoid fever of a malignant and dangerous type. The floor of the hut in which they existed was as damp and muddy as the road outside in wet weather, and as the family had not more than enough blankets to cover one bed the boys used to lie on the ground before the fire at nights.
>
> The committee of the Benevolent Society at once caused these unfortunates to be removed to the hospital, undertaking to provide them with the requisite nourishment and restoratives, and Dr. Pratt has very kindly and charitably promised to attend them gratuitously.

Such was the position of the needy poor. The wealthier sections of the community were of course able to be attended in their own homes, although even at best these were built of wattle and mud, of wood, or rarely of stone. Operations, especially for patients residing at a distance, were a worry; but Dr. Pratt surmounted this trouble by arranging accommodation in the town. An extract from a booklet he later published, a copy of which is to be found in the Sydney Mitchell Library, gives some idea of

the difficulties he had to overcome. The booklet has the following title: "Notes on Tumours and Cancers with Remarks on a new and painless method of treating them. By Eustace H. L. Pratt, M.D., Member of the Royal College of Surgeons of England &c., Late Surgeon to the Forbes and Gundagai Hospitals. Formerly Surgeon in the Army." The extract reads as follows:

> Ovarian tumour in a lady aged 34 years, operation and recovery.
>
> Mrs. S., a lady living about eight miles from Gundagai, was very much emaciated from constant suffering and mental depression and her anæmia and pallor was so great as almost entirely to destroy, for the time, the appearance of her naturally prepossessing and handsome features. She had consulted other medical men and at last placed herself in my hands for the operation. I need not give the details of the diagnosis, as I was perfectly satisfied of the nature of the tumour, as indeed had been other medical men referred to above.
>
> The lady's husband had rooms prepared for her at the well-known hotel[1] of my friend Mr. J. Leary, Gundagai; and there after all the usual preparations I operated.
>
> The contained fluid was thick and opaque, the adhesions numerous, and the operation presented more difficulties than I had anticipated. Moreover, the two nurses who had engaged to assist me with sponges, water and all the little aids so necessary in such a case both failed on seeing my first incisions into the abdomen, and had it not been for the great kindness of Mrs. J. Leary I hardly know how I should have got on, for that lady came and assisted me herself.
>
> I applied silk ligatures after the removal of the tumour, sponged out the abdominal cavity and followed the usual treatment as to dressings, etc.; but I gave a large dose of opium which is my invariable practice after a very serious operation. The case got on remarkably well, the lady being up and about in six weeks and soon regaining her flesh and good looks.

So much for the details of an operation that even now represents a procedure of magnitude. That it was successfully accomplished then, in such circumstances, was almost a miracle.

Dr. Pratt records many of his cases in the little book, which was on sale to the public at the price of one shilling and which publication no doubt brought him many worried patients. His description of a case of tumour of the jaw should be a classic. I particularly enjoyed the *joie de vivre* displayed in the surgery by the patient's father, almost certainly the fat butcher from Wagga, Tom Castro, who was to become famous as the amazing rogue of the Tichborne case.

> Miss Fanny C., the daughter of a butcher living in Wagga Wagga and Tumut. The young lady, aged 13, would have been remarkably pretty but for the frightful disfigurement of the tumour which involved the whole of the upper lip and side of the cheek and nose, and was firmly attached to the superior maxilla pushing out the side of the face and making the poor girl look perfectly hideous.
>
> The father had consulted the leading surgeons in Sydney and Melbourne as well as those of several of the inland towns, but they had all refused to remove the tumour, saying that it was too dangerous and that it involved too much of the bones of the face. He had again gone to Sydney and tried to get the child into the Sydney Infirmary, but he had been refused and told that nothing could be done.
>
> Eventually he came to me, and after I examined the tumour I was glad to be able to tell him that I would remove it and with very trifling danger to life. I never remember to have seen a more pleased man than was this poor fellow. He fairly danced about my consulting room with joy.
>
> Two months after he took a cottage in Gundagai, close to my own residence, in order to be near me, and after preparing the young lady for a few days, I operated and successfully removed the whole of the tumour, which was about the size of an orange, but with very little cutting away of the bone.
>
> The child made a splendid recovery, being able to go home with her parents five weeks after the operation, and even then her appearance had improved wonderfully, and when three months afterwards they brought the child to see me I did not know her, for she had become an exceedingly pretty girl and there was a very small scar, scarcely perceptible!

For those who may be interested, the "new and painless method" of treating cancers is well indicated in the report of one case from several in the brochure alluded to above.

> Fibroid tumour of the cheek. Treated by iodoform paste. Recovery. E. H. Esquire, a squatter, consulted me in Bathurst for a tumour of the face about the size of a walnut, which had been gradually increasing for the past two years.
>
> He wished to have it removed with the knife, but as I asked him to let me try my plan without the knife, he consented.
>
> He remained under my treatment for a fortnight (when he was called away unexpectedly by important business), when the tumour had nearly entirely gone, all that remained being a small hard lump about the size of a bean, which was completely hidden by the whisker.
>
> My treatment had consisted in the injection, daily, of iodoform, combined with its external application in the form of paste. I have since heard from this gentleman, and he tells me that he is quite satisfied that he is cured.

Dr. Pratt is careful to point out that he does not "for a moment intend my readers to imagine that *all* cases of tumour can be cured this way, but that there are *many cases* which, by judicious and skilful selection, can be cured".

In 1875 Dr. Pratt published a further booklet. The title page reads: "On the Treatment and Permanent Cure of Stricture of the Urethra, with notes of fifty cases cured by the author." It was published by C. T. Sandor, 324 George Street, Sydney. A copy is to be found in the Mitchell Library, Sydney. With regard to the cause of stricture, Dr. Pratt states his "conviction that *hard, rough riding*, so common in the bush, is the most common cause of the disease in Australia". He further states that he has tried all the text-book plans of treatment and has been satisfied with the results of none of them, the cures not being permanent. He has therefore adopted a plan "after a great deal of study and thought and many trials of different substances", which consists "in the introduction of iodoform suspended in glycerine into the urethra on *both* sides of the stricture by means of an instrument which is a modification of Lallemond's porte caustique". He declares that he is "an entire upholder of Mr. Syme's doctrine that there is *no* stricture through which an instrument cannot be passed by skill and *patience*", and goes on to add that "there are many medical men practising in the bush of Australia, to my own certain knowledge, who could hardly pass a catheter for a man with a perfectly healthy urethra, so little do they remember of anatomy or so little have they ever practised this branch of surgery". Dr. Pratt refused always to give chloroform "in any cases of stricture as I believe that the heart is always weak in these cases and in fact there are many cases on record that painfully prove this". Anent this he avers that he need "only refer to the case of poor Dr. Scard, of Sydney, who died two years ago at his house in Sydney whilst being treated for stricture *under chloroform*, by two leading Sydney surgeons". He also instances the fate of "poor Louis Napoleon, the late Emperor of the French, who in my opinion was a well meaning, honourable and good man", and who "would very probably have been alive now, had he been treated for his stricture and stone without chloroform".

The author's constant defamation of his brother medical men, examples of which have been quoted, his novel publications for general sale, and eventually his support for Professor Smith, who condemned the surgeons of the day in the Legislative Council, were soon to bring down upon him the wrath of the majority of his colleagues. *The Medical Gazette* of July, 1875, *inter alia*, made the following statement in a leading article:

> We cannot have much respect for the utterances of a member of the profession who so far forgets himself as to advertise "A new and painless method of curing

[1] The Criterion Hotel, now rebuilt, is still the freehold property of the same family today and is managed by a grandson of the Mr. J. Leary mentioned by Dr. Pratt.

tumours and cancers" (*vide Bathurst Times*, 14th July); nor can we wonder that one who has crossed the Rubicon dividing legitimate practice from quackery should associate himself with the enemies of his profession; we had thought better things of Dr. Pratt. We can only conclude that he elects to make to himself friends of the mammon of unrighteousness, and we must charitably hope they will receive him into their houses.

This was too much for Dr. Pratt. He promptly issued a second edition of his booklet on cancers and penned an addendum on the concluding page. The wording is vitriolic:

> I notice that the medical men connected with that "mutual admiration" organ, the "New South Wales Medical Gazette", have attacked me with their well-known brotherly love, because I made a speech in Professor Smith's defence at a late meeting of the professor's friends at the Chamber of Commerce, and also because I have had the audacity to publish medical works without first submitting them to these worthies.
>
> Let me tell these would-be *leaders* of better men than themselves—these critics or would-be critics, of things they are incapable of comprehending, that although I am not the happy possessor of either a *Brussels*, *Heidelberg* or *Göttingen*, no, nor even a *Philadelphian* degree (the depreciation in value of these degrees has lately been so utterly ruinous to the proprietors that I notice that the prices are not even quoted in the London and American papers), I nevertheless consider myself, and have the pleasure of knowing that I am considered by my friends and patients, as somewhat better than any of these self-satisfied nobodies.
>
> It has never yet been my misfortune to lose the life of a gentleman under the simple operation for epithelioma. Nor has anyone ever died in my hands under chloroform when he had previously begged not to have chloroform for a very simple operation.
>
> Let these gentlemen point to *one* operation that will compare to several that are mentioned in this monograph and I will consent to hear them.
>
> Let me remind those gentlemen that "those who live in glass houses should not throw stones", or if they *will* insist upon ignoring this old "saw", I can assure them that I shall be able to throw *rather heavier* metal than they can.
>
> As they are apparently fond of Latin (it looks well to appear to know a *little* Latin), I will conclude with a quotation to them:
>
> *Noli adfectare, quod tibi non est datum,*
> *Dclusa ne spes ad querelam recidat.*

The above communication was addressed from 69 William Street, Sydney, to which place Dr. Pratt had transferred his practice, having given up the rustic delights of Gundagai and Forbes only to enter the rough and tumble, and no quarter, that were to be met with in the medical world of old Sydney. He must have found the going too heavy, for he soon removed to Tamworth and from there to the quiet backwater of yesteryear, Parramatta. He died on October 17, 1899, at the age of fifty-eight years, at North Sydney.

Of his family the most famous was Ambrose Pratt, who was born to the erstwhile Caroline Kershaw, at Forbes in 1874. Ambrose Pratt[1] was to become the author of many famous novels on Australian life. He studied law, and in 1905 joined the editorial staff of the Melbourne *Age*, resigning in 1918 to edit *The Industrial and Mining Standard*.

Dr. Pratt had come a long and tiring journey since the day he first met the little schoolma'am at Gundagai. But it was a journey full of event, and one typical of that led by many a pioneering country doctor. When his span was coming to its end, who knows but that he put down his scalpel—and his pen—with a sigh of contentment. For he had bequeathed Australia a famous son.

[1] Ambrose Pratt, C.M.Z.S., Vice-President and aforetime (for fifteen years) President of the Royal Zoological Society of Victoria; member of the Zoological Board of Victoria; born 1874, died 1944; author (*inter alia*) of "David Syme" ("The Father of Protection in Australia"), "Three Years with Thunderbolt", "The Centenary History of Victoria", "Lift Up Your Eyes" (a novel), "Everyman" (a narrative poem), "The Call of the Koala", "The Lore of the Lyre-Bird", "Magical Malaya", "The Real South Africa" *et cetera*.

A PLEA FOR SIMPLER MEDICAL WRITING.

By S. F. McDONALD,
Brisbane.

THE language of medicine is becoming more and more removed from ordinary speech, and by this removal medicine itself is beginning to suffer. The faults which are converting medical writing into a jargon fall into three groups, which the following quotations will explain. The first is from Butler's "Hudibras":

> A Babylonish dialect
> Which learned pedants much affect—
> 'Twas English cut on Greek or Latin.
> Like fustian heretofore on satin.

Jim Pinkerton, in R. L. Stevenson's "The Wrecker", says:

> That's a good catching phrase, hebdomadary, though it's hard to say. I made a note of it when I was looking in the dictionary how to spell *hectagonal*. "Well, you're a boss word", I said; "before you're very much older I shall have you in type as long as yourself. And here it is you see."

Mark Twain, in "A Tramp Abroad", has the following sharp conversation with his agent Harris:

> "What does hogglebumgullup mean?"
> "That is the Chinese for 'weather'."
> "Is hogglebumgullup better than the English word? Is it any more descriptive?"
> "No, it means just the same."
> ". . . Then why do you use them? Why have you used all this Chinese and Choctaw and Zulu rubbish?"
> "Because I didn't know any French but two or three words, and I didn't know any Latin or Greek at all."

Today the medical student knows little Latin and less Greek; however, unlike Harris, he is not allowed to take refuge in Chinese or Choctaw or Zulu, but he is given massive lumps of Greek and French or Latin and German and is expected to find out what they mean—if he likes. Mostly he does not, but he goes on repeating words like *débridement* (though he leaves out the accent and stresses the wrong syllable) and talks about kernicterus or *erythroblastosis fatalis* in the free fashion of a man who has all the tongues of Pentecost. Ask the average doctor what *débridement* means, and he will tell you much—but it won't be quite what Larousse says. (*"Petit Larousse"* is the miniature encyclopædia which every French child has when he starts work at his secondary school.) I have just been trying to write a short lecture on the problems of the Rh factor, in simple language, that could be easily delivered and (I hope) easily understood. Apart from such more or less necessary atrocities like heterozygote and allelomorphs, the papers on the subject are full of such classical satin as *hydrops fatalis* and *icterus gravis neonatorum*, or such "boss words" as "erythronoclastic". (It was most refreshing as a contrast to find the admirable article of Henry and Simmons in THE MEDICAL JOURNAL OF AUSTRALIA of June 29, 1946, written in simple, straightforward English.) I have not come across "alibi" used instead of "excuse" (which it does not mean) in any recent papers, though it was so used in an article in this journal some years ago.

So much, I think, for the English "cut on Greek or Latin" group. Let us now turn to some "boss word" usage. A phrase which seems to have arisen during the war is the radiologist's stock report: "No osseous lesion detected." Three of the words here are "boss words", and apparently preferred in the way that Pinkerton preferred "hebdomadary" to "weekly". "Osseous"! Why not "bony"? "Lesion" is a word slowly making its way into medical English as a general utility word for "fracture, wound, disease, congenital deformity". For "injury" it is right enough; "*lædo*", the Latin root, means "I hurt"; by a stretch it may include disease, but I cannot see it fairly used in any other sense. And "detected": the radiologist does not detect; Mr. Sherlock Holmes did that. One may permit the word to the busy house surgeon when he writes "N.A.D.", because he has applied all his senses (not, I suppose, taste, now that today we have Benedict's

solution) in examining a patient. But the radiologist has one sense only that he can use in interpreting his pictures: sight. So he should write in his report "No bony" (or "joint", or "lung", as the case may be) "change" (or "abnormality" we may allow him as a treat) "seen". Also this will make life easier for the poor typist who has to translate a scrawl into fair typescript.

There is another and most diverting form of the "boss word" when one has a taboo or prudery complex mixed with it. Take that dreadful phrase now so popular: "voided urine". "Void" as a verb is a translation of the French "*vider*", which means "to empty". So actually "voided urine" means "emptied urine", which is, I suppose, possible, though it sounds untidy. The French phrase which is perfectly translatable is "*vider la vessie*"—"empty the bladder". (Falstaff sang "Empty the jordan".) But about the original English phrase "passed urine" there is a vague hint of vulgarity, "something not quite nice"; and as this especially appeals to a certain type of woman, nurses and sisters have taken up "voided" with great relish—sometimes making the transitive verb intransitive by simply saying "he voided". (So great is this horror of calling a spade a spade that I know one hospital where the nurses are forbidden to speak of squares, napkins, diapers; they must say "sundries". I am told that to use the phrase "all and sundry" in the wards is to make the nurses as uncomfortable as the Boston lady who heard the word "leg" in public.) Actually I do not think that the taboo words matter so much (Robert Graves wrote a most amusing little book on the matter some years ago) when they do not conflict with actual medical fact, but there is one very bad offender in this group. I refer to "gastro-enteritis", especially the "gastro-enteritis" of small children. Many years ago, when I was his house physician, each morning we had the dismally familiar sight of Dr. G. Douglas Stephens doing post-mortem examinations on these little victims at the Melbourne Children's Hospital. There were many of them; some showed no sign of inflammation from gullet to anus; the others showed inflammation of the large bowel only. I can remember Dr. Stephens's excitement on the morning when he found an obvious area of ulceration two feet above the ileo-caecal valve; but he never found any abnormality in the stomach. I suppose the disease was called gastro-enteritis because vomiting is often an initial or even an almost constant feature. Vomiting also occurs in appendicitis in children, but no one talks about "gastro-appendicitis". Pneumonia is often ushered in by vomiting; I have yet to hear of "gastro-pneumonia". But the word "gastro-enteritis", usually shortened down to "gasteritis", has been taken over by the laity, and you will be told that "the child had the gasteritis" or "the motions were very gastric"; suggest that the baby really had diarrhœa and watch the reaction. I can understand "purge" being dropped; it is an old-fashioned word and one now used to mean "a massacre of political opponents"; but why a fine resonant Greek word like "diarrhœa" should be discarded I cannot understand.

Now my objection here is not merely an Herbertian interest in the accurate use of words as words, but because of the ideas which words carry. Give a student (or a practitioner) the idea that the summer diarrhœa of infants is something to do with their stomachs, then the idea that the vomiting may be due to a body fluid disturbance or to an upset of the bowel gradient will never thereafter come into his mind and he will do all sorts of curious things to relieve the stomach disease (usually giving bismuth), instead of securing relief of the dehydration and some treatment for the ulceration in the last few feet of large intestine.

But the latest example of medical "literary pomposity" I have seen is the new art of anæsthesiology (and its practitioners anæsthesiologists). What a word!

Another form of verbal misuse in medicine and surgery is exactly like Harris's. He wanted to make his report impressive, so he put in as many foreign words as he could think of, regardless whether there was any need to do so and whether or not the English equivalent was not quite as expressive. Often people who do this will tell you that there is no exact equivalent in English. Often this is quite true—speculum and *pébrine* and *kernschwarz* are examples; but there are not so many as people would have us believe. I still remember how I failed to convince my French master that there was no English equivalent of "chiffon". They order this matter better in France: a foreign word, especially a Latin or Greek word, must assume a French dress and gender if it is to be freely used and understood by French workers; especially is this the case in anatomy. I do not know whether the French have tolerated the wholesale Latin of the *Basle nomina anatomica*, but certainly "*patelle*" does not mean the patella.

There is little need for such words as kernicterus, *Zwieback*, *Eiweiss Milch*, *débridement* and *tulle gras*, which are trying to find a home in English, to the great discomfort of the student and the delight of those who prefer to use them rather than the equally effective and much more intelligible words of our mother tongue.

Let a master state the case better than I can ever do (Mark Twain speaking to Harris):

> The writer would say he only uses the foreign language where the delicacy of his point cannot be conveyed in English. Very well, then he writes his best things for the tenth man, and he ought to warn the other nine not to buy his book. However, the excuse he offers is at least an excuse; but there is another set of men who are like you; they know a word here and there, of a foreign language, or a few beggarly little three-word phrases, filched from the back of the dictionary, and these they are continuously peppering into their literature, with a pretence of knowing that language—what excuse can they offer? The foreign words and phrases which they use have their exact equivalents in a nobler language—English; yet they think they "adorn their page" when they say Strasse for street, and Bahnhof for railway station, and so on—flaunting these fluttering rags of poverty in the reader's face, and imagining he will be ass enough to take them for the sign of untold riches held in reserve.

But there is still another reason to avoid such tricks, and that is that simple English is more direct and forceful in the hands of the ordinary writer. Dr. Johnson and Gibbon could write in great rolling periods and words of classic origin; but take Huxley's lecture "On a Piece of Chalk" delivered to working men and try to rewrite it in Johnsonese. Take an extreme case. As a child I had a much prized copy of "Sandford and Merton" in words of one syllable. I read it and reread it and today I remember it as an excellent, directly told story; the episode of the bull-baiting is particularly vivid. I have just tried to reread it in Mr. Thomas Day's own words; it is a smug, tedious tale, that I can imagine no child tolerating for a moment.

At present there is much talk of Basic English and its value among primitive peoples. Although I think the founders of Basic English have tried to be too simple and that educated men and women may be expected to rise to something a little better and more direct, yet there is an opening for a much simplified style in our medical writing. This aspect of the whole matter, I consider, cannot be too much emphasized. Medicine is becoming more and more difficult as the years go on, and lack of lucidity is the greatest bar to learning and understandiag. Walter Murdoch in his essay an "Sesquipedalianism" points out that clear writing is always simple writing.

> You are impressed by that kind of writing; you know you are. You may not admit, consciously, that long words are a sign of what is called "culture", but you do, somehow, conceive a greater respect for a writer who writes "over your head" than for one who is easily and immediately understandable.
>
> You would be reluctant to pay a fee to a doctor who told you that you had got the hump; to satisfy you, he has to say you are undergoing an attack of neurasthenia; yet it means just the same. The general rule holds good; long words are a sign either of muddled thinking or of sham erudition.
>
> This attitude towards words is, I say, wrong. When a man who uses our English speech is thinking clearly

he does not, in actual fact, think in long words, but in short and plain ones. And the good writer is he who writes as he thinks.

Whether we should try to do more to educate our medical students to write I do not know; but it is certainly depressing at present to find how few can sit down and write a short essay (which is what our final examination question should be) in lucid nervous English. Assuredly they are not being helped by the jargon which their teachers and would-be teachers adopt.

Let us again consider two masters. What was one of the factors that made "Osler" the medical Bible of a generation of students? The clearness and directness of the language. Osler's own article on "Typhoid" is perfect and easy-running English, though actually I should put as the finest of all his writing the passage in "*Æquanimitas*" beginning: "Indeed", as Saint Paul says, "it is better to marry than to burn." Take another master's work—Sir Robert Hutchison's lecture on the "Chronic Abdomen"; it is compounded of shrewd common sense, perfect English and profound clinical understanding, anticipating most of the "psychosomatic" teaching by twenty years. If you object that this is a "lecture" and so must needs be simple, take any article by Mackenzie or Lewis on heart disease and see if you can better them.

Such clear and direct teaching makes the study of medicine much easier; this article is written in the hope that those responsible for the teaching of students and for the setting out of new knowledge may discard some of their present jargon and come back to a language more easily understanded of the people.

POLYCHLORNAPHTHALENES AND LIVER NECROSIS.[1]

By J. M. DWYER,
Adelaide.

IT is believed that these cases are the first of their type to be reported in Australia, although references to the condition may be found in the world's literature, mainly since 1933. Cases of a similar origin occurred in Germany during the first World War from the making of perchlornaphthalene for synthetic rubber.

Case I.

A patient, aged thirty-five years, was admitted to hospital on November 10, 1944. For a month previously he had suffered from indigestion, distension of the upper part of the abdomen, belching, heartburn and sour fluid in the throat, and nausea and vomiting had occurred about twice a week. He had lost his appetite over the last month. Three days before his admission to hospital he had had a severe attack of vomiting, during which he brought up brown fluid. Next day he was noticed to be yellow in colour. The vomiting had continued. He was constipated, and had noticed that his stools were pale. His weight was about normal at 155 pounds. Micturition was more frequent than usual in the day (about eight or nine times), but not at night. The urine had been noticed to be darker than usual. Headaches across the forehead had been troubling the patient. He had had no previous illnesses except measles at the age of twelve years, and some arthritis since August, 1942. One brother was alive and well, his mother suffered from asthma, his father had died of cancer. Before his military service he was a labourer. He took alcohol occasionally and smoked about three ounces of tobacco a week. The date of enlistment was December 13, 1941.

On physical examination, the patient was found to be well nourished. The conjunctivæ were yellow. The pupils were equal and reacted to light. He wore full upper and lower dentures. The throat, lungs and heart were normal. The blood pressure was 110 millimetres of mercury (systolic) and 70 (diastolic). The liver was tender and palpable four fingers' breadth below the costal margin. No other viscus or masses were palpable. Some crepitus and grating were heard on movement of the right knee, which could be flexed to about 100°. The reflexes were equal and active, the plantar reflexes being flexor in type. The urine appeared normal on microscopic examination, but contained bile salts. No bile pigment was detected.

The jaundice gradually increased, and indigestion continued for the rest of the month. On December 4 the patient was miserable, with no definite complaints; but the next day he became irrational and non-cooperative. His blood pressure was 125 millimetres of mercury (systolic) and 85 (diastolic); it had risen since his admission to hospital. The liver was no longer palpable. No sugar or albumin was found in the urine. The pupils were dilated. Lumbar puncture revealed normal cerebrospinal fluid. The senior physician at this time recorded his view that the condition was one of liver necrosis.

During the next three days the patient became restless and unable to answer simple questions. He began to vomit, and his condition deteriorated. The temperature began to rise on the second day, and the restlessness increased for a time, but he became quiet later at night. The next morning he became comatose and died about 11 o'clock.

Post-Mortem Examination.

A post-mortem examination was made. The body was that of a well-built man, aged about thirty years, with pronounced generalized jaundice. Post-mortem staining and rigidity were present. The lungs contained air and showed a slight degree of anthracosis. Adhesions were present between the inner surface of the right lung and the mediastinal area, and also between the base of the lung and the diaphragm. Both lungs were congested. From a small area in the left lung a few small beads of purulent material were expressed.

Several small hæmorrhagic spots were found on the posterior surface of the heart. A yellowish discoloration was noted in the aorta and the tissues of the heart. The heart muscle was soft.

Free fluid, yellowish in colour, was present in the abdominal cavity. The liver was about half normal size, with raised nodules, yellowish in colour and of varying size, scattered over the liver surface, mainly the lower half. In between the raised patches the liver substance was bright red; the appearance was most striking. There were omental adhesions to the gall-bladder and the first six inches of the ascending colon, the wall of which was thickened and hæmorrhagic. The pelvic colon had a similar appearance. The bile-stained and congested stomach mucosa was covered with thick mucus. Hæmorrhage had occurred near the cardia. The spleen was very soft and congested. The kidneys were larger than normal and congested. The left cortex was not so easily distinguished as the right. All internal organs were heavily bile-stained. A small, hard gland in the left groin was removed for the preparation of sections.

In the microscopic sections that were prepared from the subject, some degree of collapse was evident in the portion of the left lung from which the pus had been expelled. In the liver an advanced stage of portal (multilobular) cirrhosis was present. Chronic inflammatory changes and œdema were seen in the lymph gland. The congested areas in the large intestine were due to post-mortem changes only. Death was attributed to acute yellow atrophy of the liver.

Case II.

Another patient possibly suffering from the same disease was admitted to hospital on January 10, 1945. He had been working with chlorinated naphthalene for seven and a half months. In the last four months he had become constipated for as long as three days at a time. Loss of appetite had occurred, and about six weeks previously he had vomited two or three times. He had lost about eight pounds in weight and was troubled by frequent headaches. He affirmed that he always washed his hands before eating or smoking. The bath in which the chlornaphthalene was heated had no thermometer, and the heating was done with "Primus" burners, which were difficult to control. The chemical often bubbled a lot. Besides this

[1] Read at a meeting of the Australian and New Zealand Association for the Advancement of Science, Adelaide, August, 1946.

fumes came off the treated metal while it was being allowed to cool inside the shop before the electrodeposition was begun.

On the patient's admission to hospital, his appetite was noticed to be poor and he was costive. Three days previously he had noticed that his motions were grey in colour. No change was noticed in his urine, and he had no frequency of micturition. He had a cough with sputum, but no hæmoptysis, and no undue shortness of breath. He had suffered from pneumonia and scarlet fever. His family history was good, his parents and siblings all being alive and well. He did not take alcohol, but smoked about three ounces of tobacco a week. His weight was normally 129 pounds, but on his admission to hospital it was 121 pounds. General examination gave entirely normal results, except for tenderness to the left of the umbilicus.

Because of the previous case the patient was sent to Melbourne for a liver function test. On his arrival there a heavy white fur was noticed on his tongue, and the abdominal tenderness had disappeared. The test was performed on January 17, 3·48 grammes of benzoic acid being excreted in four hours. This figure was normal, as also was the albumin-globulin ratio (4·85:2·4). He returned to us on February 1 and was discharged fit to his unit a fortnight later.

Discussion.

I was present at the post-mortem examination on the first subject (Case I), and considered it worth while checking on the type of work done by the deceased, as it appeared to me that some industrial toxin might have been responsible. At first I thought the illness might have been due to some substance like tetrachlorethane. It was found that the patient had worked with the following: the electrical deposition of metals, trichlorethylene, range fuel, polychlornaphthalene, chromic acid, nickel sulphate, sulphuric acid, and cyanide (occasionally). At first the search was concentrated on the possible culpability of trichlorethylene, but I recollected reading the paper by Peck[5] a few months earlier. It was stated that the patient had worked most with the chlorinated naphthalene wax, but prior to this had worked with the trichlorethylene in the degreasing vats. The significance of this was not apparent till later. An inspection was made at the workshops where he had worked. Not far from the chromium plating room where the "Halowax" ("Seekay" wax or polychlornaphthalene) was used were the containers of the trichlorethylene, the chloroform-like odour from which could be readily detected. Near the door of the chromium deposition department the curiously intense, pungent smell of the vapourised "Seekay" wax could be smelt—one might almost say, felt. The immersion tank was being heated by pressure burners and the fumes could be appreciated as one approached. After a short time one became conscious of the fact the odour was less intense, though the concentration was in fact unaltered. Collections of the sublimated wax could be taken off beams and cross-supports literally in handfuls. An exhaust fan was installed, but it was too far from the focus of dissipation of the fumes and the quenching troughs to produce an effective draught. It is not known what the actual concentration of the vapourized wax was at this time, but the permissible limits will be discussed later.

The clinical report of this case is not detailed, but there is a close correlation with the description given of chlorinated naphthalene poisoning by McLetchie and Robertson,[1] Collier[2] and Cotter.[3] The absence of acne on the extensor aspects of the forearms and face is notable in this case; but in the case described by McLetchie and Robertson and other writers it was absent. Members of the patient's unit averred that he had had acne, and that there were two others working in the same shop who suffered likewise. As those concerned had moved on to other postings, it was not possible to verify the statements. Because of this, and because of the lapse of time and the uncertainty of opinion, the statements may be unreliable. A similar statement was made with regard to the feeling of revulsion for the work in the shop because of the smell. During the inspection men working expressed their distaste for the work; but one gained the impression that the job was unpopular and under suspicion because of the death of their fellow worker, who had been noticed to be "out of sorts" for some time before going to hospital.

On perusing the literature one is struck by the occasional mention of previous rheumatic trouble. This association may be accidental, but it appears to be more frequent than would normally be expected in a group of factory workers mainly aged below fifty years. Collier suggests that damage to the heart may have affected the liver in his case, but apparently does not otherwise implicate the attack of acute rheumatism nine years previously. The dilated pupils and frequency of micturition present in this case have been referred to in reports.

The post-mortem examination in the case described by Johnstone[4] provides a vivid description of the appearance of the liver in the present case, and the illustrations in Cotter's article bear a close resemblance to the microscopic appearance of sections taken from the specimen of the patient's liver.

There are two types of tissue. One, containing little liver parenchyma, was yellowish at the post-mortem examination, and the other, which contains abundant fibrous tissue enmeshing many leucocytes, blood corpuscles and vestiges of bile ducts or islands of liver parenchyma, was bright red. The generally accepted view that the islands of epithelial tissue remaining in the fibrous tissue are bile canaliculæ is opposed to the view that these patches may be in reality islands of liver cells squeezed and atrophied in the fibrous tissue. Eosinophile cells, which are recorded in the degenerated liver sections in Cotter's article, are present in the sections from my case. With this profound degeneration it is easy to imagine the nobby appearance of the liver.

Points in Prophylaxis.

Reports indicate in places that some persons are more susceptible to this type of damage than others. On this account it is thought wise to exclude those who are possible subjects of liver damage from working with these substances. Therefore, those who have had infectious hepatitis, jaundice, malaria or typhoid fever, or those who are pregnant, should be debarred. Obviously those who have had "Avertin" or chloroform anæsthesia may be affected. The converse association should be borne in mind, and because of the damage recorded by trichlorethylene and tetrachlorethylene, workers in these substances should not be put to work with "Seekay" wax or similar substances. The possible association in this case is perhaps important. Industrially it is probable that the two processes may be carried on in adjacent sections of the same factory, as in this case. Similarly, one should perhaps remember not to put the chrome-platers back on to the degreasing processes with the workers with trichlorethylene. Writers appear fairly unanimous that there is no suitable test to indicate the presence of liver damage in these cases, and agree that there is no substitute for clinical observation and examination. The presence of fumes of the molten substance especially with over-heating should serve as a warning. Both these factors were present in this case, and visiting inspectors agreed on the existence of the risk. The permissible limit of pentachlornaphthalene concentration is generally stated to be 0·5 milligramme per cubic metre. Higher chlorination makes for greater toxicity. After the installation of a much improved draught ventilation, a figure of 0·15 milligramme per cubic metre was obtained above the waxing bath. The average diameter of particles was of the order of 0·8μ.

Summary.

A case characterized by signs and symptoms consistent with polychlornaphthalene poisoning is described. The post-mortem findings and pathological histology support the clinical aspects. It is believed that this case is the first of its kind reported in Australia.

Some methods of control are mentioned.

Acknowledgements.

I am indebted to Dr. D. O. Shiels, Medical Officer for Industrial Hygiene, Melbourne, for verification of my views and for supplying a bibliography of relevant work, to Dr. W. R. Moloney for the clinical notes of the first case, and to Dr. Eileen Cammack for the post-mortem notes.

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A NOTE ON OGILVIE'S TUBULAR DIRECT HERNIA.

By C. CRAIG, M.D., M.S., F.R.A.C.S.,
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IN Maingot's "Post-Graduate Surgery", published in 1937, W. H. Ogilvie described a rare form of direct hernia, "in which the posterior wall of the inguinal canal medial to the deep epigastric artery shows a small circular deficiency with firm, almost tendinous margins, through which a tubular process of peritoneum, indistinguishable on clinical examination from an indirect inguinal hernia, escapes and often emerges through the external ring". The writer has seen six such herniæ. The purpose of this note is to put on record certain anatomical details associated with them.

Position.

Two herniæ were close to the pubic spine. The other four came through the posterior wall about halfway between the inferior epigastric artery and the pubic spine.

Insertion of Conjoined Muscle.

In each case the insertion of the conjoined muscle was very high, so that the lowest point of the insertion was at least 2·5 centimetres (one inch) above the pubic bone. Because of this high insertion, a triangular area between the lower border of the conjoined muscle and Poupart's ligament was completely uncovered by muscle when the external oblique had been reflected.

Fascia.

The fascia in this region varies greatly. Although usually of an areolar type, it is occasionally much tougher. In all these cases it was exceptionally tough, and approached in texture that of the aponeurosis of the external oblique.

Comment.

It seems possible that these herniæ are "pulsion diverticula" forcing their way through a weak spot in fascia which is unprotected by muscle.

Reports of Cases.

CHROMOBLASTOMYCOSIS: REPORT OF A CASE.

By J. D. HICKS,
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Definition and Ætiology.

CHROMOBLASTOMYCOSIS is the name given to a chronic granuloma of the skin, caused by a group of fungi, of which the characteristic feature is that in the tissues the double-contoured organisms have a deep yellow or brown colour. The prefix "chromo" distinguishes the condition from that caused by the colourless organism of blastomycosis. Moore and his associates[1] insist that the disease be called chromomycosis, as the fungi producing these lesions are not blastomycetes; budding forms are not found in the tissues, and division appears to take place by septum formation. Several closely related varieties of fungus have been identified by artificial culture.[2] *Phialophora verrucosa*, *Hormodendrum pedrosi* and *Hormodendrum compactum* are the species most commonly isolated.

Occurrence.

Chromoblastomycosis is found chiefly amongst farmers. It is suggested that the fungi occur on vegetation and are occasionally inoculated into the tissues by trauma. The condition is well known in tropical America, especially in the Amazon Valley, but has been uncommon in the United States, only ten cases being reported up to 1944.[2] It has, however, a wide geographical distribution, cases being found in the Latin American States, in Japan, in Java, in Russia, and in various regions in Africa.[3][4] Saxton, Hatcher and Derrick[5] have reported two cases in Australia; a third is presented in this record.

Clinical Record.

The following case report is of special interest, in that the lesion developed while the patient, a soldier, aged forty-two years, was stationed in the Northern Territory. He was in a small "Commando" group travelling about the Cambridge Gulf and visiting small islands off the coast of Australia for two and a half years. He frequently lived in close contact with the Myall blacks for months at a time, and in some camps were lepers and tuberculous patients who had escaped from the Darwin colony at the time of the air-raids.

The lesion was first noticed fifteen months before it was removed. A purplish, slightly raised patch developed over the medial condyle of the right femur. It was irritable and was continually scaling, but it never broke down or discharged. When it was completely excised by Major A. J. Foote, on June 19, 1945, the actual nodule was oval in shape, measuring 1·25 by 0·75 centimetres, and rose slightly above the level of the surrounding skin. A whitish appearance near the margin of the nodule suggested a hyperkeratosis.

Histological Findings.

Microscopic examination revealed considerable hyperplasia of the epithelium (Figure I), with irregular protrusions into the dermis giving the appearance of a squamous cell carcinoma. Within the epithelial masses were small abscesses containing degenerating polymorphonuclear cells and debris. The dermis beneath and between the infiltrating epithelium was packed with cells, plasma cells and polymorphonuclear cells predominating. Small and large round cells were numerous, and Russell's acidophile bodies were frequently seen. In a dozen places epithelioid cells were grouped, often with central polymorphonuclear cells, into small pseudotubercles, with or without giant cells of the Langhans type. These last-mentioned cells were also scattered amongst the plasma cells. No caseation was present, but often in the centre of these tubercles was to be found a highly refractile cell, circular or sometimes convoluted in outline. It was about 10μ in diameter, had a thick double-contoured wall and was yellow or brown in colour. Occasionally a small group of the cells appeared to have been formed by septate division (Figure II).

Comment.

As in this case, in which the whole lesion was fixed in formalin, diagnosis may rest on the discovery of the characteristic yellow double-contoured spores in a histological preparation. The spores may also be discovered in pus or in scrapings, and isolation of the fungus by culture is necessary to its subsequent specific identification. In most cases the organisms are strewn profusely through the tissues, but in this case the maximum number found in one section was nine, and in some sections only three were counted during careful search of the whole section with the high power of the microscope. It is therefore possible for a number of earlier nodules to be excised and for a less exact study of the sections to fail to reveal the true nature of the condition.

We are unlikely in Australia to see the gross hypertrophic warty lesions such as "mossy foot", endured for as long as twenty years by the peasants of South America; but we shall be confronted more often with a small,

erythematous, indurated or nodular area which is scaling, or is ulcerated, of months' rather than of years' duration.

It has been found in America that in some cases a good response has followed the exhibition of iodides. In early cases surgical excision may be the best method of treatment; but in advanced cases cure appears to be difficult. However, the disease remains confined to skin and subcutaneous tissues and it does not seem to affect the general health of the patient.

Summary.

A third case of chromoblastomycosis occurring in Australia is reported. A brief outline of the condition is given, and attention is drawn to the relevant literature.

Acknowledgements.

My thanks are due to Major A. J. Foote, of an Australian Camp Hospital, for his kindness in supplying the clinical data concerning the material he sent for examination. I am indebted to Mr. Woodward-Smith for the photomicrographs. The Director-General of Medical Services, Australian Military Forces, Major-General S. R. Burston, has kindly given permission to publish this case report.

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A CASE OF RETICULO-SARCOMA.

By EDGAR STEPHEN and DOUGLAS REYE,
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TUMOURS of the reticulo-endothelial system still provide much ground for controversy, and no entirely satisfactory classification for these processes has been evolved, nor is there any general agreement between what constitutes hyperplasia in contrast to neoplasia in this group. In a recent publication Gall and Mallory[1] have presented a simplified classification for some of these conditions under the title of "malignant lymphoma", and they have endeavoured to correlate the pathological changes with the clinical picture. Robb-Smith,[2] on the other hand, has suggested a much more intricate and detailed classification based on cytology, although in conclusion he states that further subdivision may be necessary, or certain of the classifications he has suggested may prove superfluous.

The following report may serve to show some of the difficulties in clinical diagnosis which are likely to be encountered in cases of this kind, and it does serve to emphasize the need for caution in histological diagnosis and the impossibility of applying a too rigid classification to tumours of this system.

Clinical Record.

The patient was a girl aged twelve years, with a record of particularly good health. Tonsillectomy was perfomed early in July, 1944; she made a good revovery and returned to school.

A high temperature developed four weeks after the date of her operation, and she was admitted to a private hospital, where she remained for twenty-six days. Dyspnœa and stridor developed; on one or two occasions the advisability of performing a tracheotomy was considered. Enlarged glands appeared under the mandible on the right and left sides, and in the preauricular region of the right side. There was a temporary enlargement of the spleen. During her stay in this hospital she was given a course of penicillin and also several sulphonamide preparations, without relief to temperature or signs.

To enable her to receive treatment with steam, she was admitted as a private patient under the care of one of us (E.S.) at the Royal Alexandra Hospital for Children, on August 26, 1944. On her admission to hospital she had stridor and dyspnœa, and her temperature was elevated to 101° F. There was a rash on the trunk resembling that seen after a sulphonamide drug had been taken. She had a thick, purulent nasal discharge which persisted, and there were noticably enlarged glands in the neck, on both sides under the mandible, in the pre-auricular regions and in the right axilla. Slightly tender swellings were present at the outer angle of the orbits. The spleen was not palpable. In the course of ten days the stridor disappeared; considerable expectoration of a clear frothy fluid occurred. The glands became reduced in size, but a group of glands in the left inguinal region became considerably enlarged. These returned to normal in the course of a week. There was an indolent area in the right tonsillar bed which persisted for two weeks. Despite the variations in signs, the temperature remained elevated, often reaching to 103° F. at some period of the day and seldom falling below 100° F., and this persisted for the twelve weeks of the illness.

Two skiagrams were taken of the chest, one on September 4, 1944, and this was reported as revealing slight chronic bronchitic signs; the other on September 25, revealed enlarged hilar glands. A blood count performed on August 28, revealed a moderate degree of normocytic orthochromic anæmia; the number of leucocytes was within normal limits. No alteration in the number of leucocytes was found in subsequent blood counts, but the number of red blood cells and the hæmoglobin level continued to fall, a blood transfusion being necessary on September 17. Many other laboratory investigations were undertaken, but nothing significant was found at any of them until a biopsy examination was made of an enlarged lymph node which had appeared on the flexor aspect of the right forearm on September 26. It was a notable feature that no complaint of pain was made at any time, and there was little disturbance from coughing, though inspiratory rhonchi persisted throughout both lungs. The blood transfusion and a course of sulphadiazine had effected no improvement, and the patient's condition steadily deteriorated until death occurred on October 16.

Autopsy.

The body was that of a tall, well-nourished girl of almost adult proportions. The feet were swollen with œdema. Permission was obtained to open the thorax and abdomen.

Thorax.

Each pleural cavity contained a considerable quantity of clear, straw-coloured fluid. The lymph nodes of the mediastinum were considerably enlarged, particularly those situated at the main bronchial bifurcation and those in the hilar region of each lung. These enlarged nodes varied in size, the largest measuring 3·7 centimetres in diameter. The smaller nodes were firm and the cut surface was uniformly white; but the larger nodes felt soft and the cut surface was mottled in white, red and yellow.

The region of the thymus was occupied by a thin layer of fibrous tissue, which contained, in some parts, notably in the right lateral border and at the base, rounded nodules, some of which felt cystic, and these were found to contain a soft gelatinous material. Enlarged lymph nodes were also found in the lower part of the neck, on either side of the thyreoid gland. The upper part of the neck was not examined.

The lungs were patrly collapsed and œdematous. Narrow white lines, which were not raised above the surface, could be seen through the visceral pleura covering the lower lobes.

The heart was much enlarged, and this was due to a dilatation of all the chambers; the muscle was pale and flabby.

Abdomen.

The peritoneal sac contained a large quantity of clear fluid. An enlarged lymph node was found in the abdominal wall, in the mid-line just below the xiphisternum. The retro-peritoneal lymph nodes were enlarged and the abdominal aorta was surrounded by numbers of these enlarged nodes. Large lymph nodes were also found surrounding the pancreas and in the portal fissure.

The spleen was only slightly enlarged and weighed 231 grammes. The capsule was smooth and not increased in thickness. The pulp was soft, moist and deep red in colour; no Malpighian bodies could be seen.

The other abdominal organs were not abnormal, and there was no increase in lymphoid tissue in the alimentary tract.

Lymph nodes removed from the inguinal region were slightly enlarged.

Pathological Histology.

The lymph node removed from the right forearm at biopsy presented none of the architectural features of a lymph node, nor did it contain any lymphocytes. It was composed of large, pale cells arranged in trabeculæ, these trabeculæ being bound by a few fine collagen fibres, and this arrangement will be appreciated by reference to Figure I, which also shows the morphology of the cells. They will be seen to be fairly uniform in size and shape; the nucleus is round to oval with a sharply defined nuclear membrane and moderately chromatic, and the majority contain one or sometimes two nucleoli. The cytoplasm is pale and abundant and in most instances is divided from nearby cells by a well-defined cell border. This cytoplasm was finely granular and faintly basophilic, though these features cannot be recognized in the photomicrograph. There were numerous mitotic figures.

The examination of lymph nodes removed at autopsy gave the following findings.

In the larger mediastinal and lower cervical lymph nodes there were many areas of hæmorrhage and necrosis; in the smaller nodes portions of the lymphoid tissue remained and the nodal architecture was retained in these parts. Apart from such variations, all the nodes examined revealed an essentially similar histological picture (see Figures II and III). The tissue was composed partly of large cells with hyperchromatic nuclei, many of them multinucleated, and partly of smaller cells which resembled the larger variety in all respects except size; the cytoplasm of these cells was opaque and eosinophilic. There were also present many cells which presented the appearances of histiocytes. They had abundant pale cytoplasm and rounded, faintly-staining nuclei in contrast to the hyperchromatic cells above described, and two cells of this type are to be seen towards the lower left corner of Figure III; lymphocytes occurred in varying numbers and an occasional eosinophile leucocyte was seen. Penetration of the capsule of the node by this tissue had occurred in many parts, and numbers of the perinodal lymphatics were packed with tumour cells. Fibrosis was not a feature, though in some nodes an overgrowth of fibrous stroma had occurred, and in occasional nodes this was pronounced.

The changes in the enlarged retroperitoneal lymph nodes were essentially similar to those found in the lymph nodes from the neck and mediastinum. The enlargement of the lymph node taken from the abdominal wall, however, was not occasioned by newgrowth, but was due to œdema, and the greatly dilated sinuses contained abundant histiocytes. This type of pathological change had also occasioned the enlargement found in the inguinal nodes, except that in these an occasional tumour giant cell could be found among the histiocytes in the sinuses, and between the lymphoid cells of the medulla.

Examination of the lungs revealed that the white lines noted beneath the visceral pleura at autopsy were caused by permeation of the lymphatics by tumour cells, and though the giant cell variety were most conspicuous, all types of cells seen in the lymph node could be found in these channels. The lymphatics surrounding the bronchi and those in the interlobular septa were permeated in a similar manner, and the connective tissue of these septa was liberally infiltrated by histiocytes and lymphocytes, though only occasional hyperchromatic tumour cells were to be found in this situation.

There was no active tumour tissue to be found in the spleen, though the Malpighian bodies were ill-defined and many were entirely replaced by fibrous tissue. The sinuses were packed with red blood cells and histiocytes, and the majority of the latter cells contained red blood cells engulfed within their cytoplasm.

The heart, liver, kidneys, adrenal glands, pancreas and alimentary tract were not involved.

Discussion.

The explanation of many of the clinical signs is evident from the autopsy findings, though the high and persistently elevated temperature can hardly be explained on the basis of the degree of necrosis found in the tumour tissue. The rapid fluctuation in size of some of the lymph nodes, particularly those in the inguinal region, which had proved a rather puzzling feature, would seem to have been due to the fact that the increase in size of these nodes was caused by œdema and sinus catarrh and not by newgrowth.

The exact histiological diagnosis, however, was not so evident. The histopathological findings in the tissue removed at biopsy were compatible with the diagnosis of reticulo-sarcoma of the type referred to by Ewing[3] as endothelioma of the lymph nodes, and classified by Robb-Smith[2] as trabecular reticulo-sarcoma, while in the nomenclature of Gall and Mallory[1] it would seem to agree with their description of a stem-cell lymphoma. It was not possible, however, to make an absolute diagnosis of reticulo-sarcoma on the purely morphological evidence presented by the biopsy material, since it is generally agreed that it is difficult indeed to differentiate this type of primary growth in the reticulo-endothelial system from secondary carcinomatous deposits in lymph nodes; and though certain associated features may be helpful in individual cases, the knowledge of the presence or absence of a primary carcinoma is often the only sure guide to the correct diagnosis. The most that could be said in this particular case was that the patient was suffering from a highly malignant neoplasm, which was likely in this instance, in view of the age period, to have its source in lymphoid tissue, but that a primary carcinoma could not be definitely excluded. Any doubt of this kind was excluded at the autopsy; but a further difficulty was presented, and this was one of the most interesting features of this case, inasmuch as in the lymphoid tissue obtained at autopsy the histopathological findings differed entirely from those seen in the biopsy material. In this autopsy material the cells were not arranged in trabeculæ, all resemblance to an epithelial newgrowth was lacking, and the tissue was characterized by the marked pleomorphism of its cells. It is tumours of this type which have been classified as Hodgkin's sarcoma, while the name suggested by Robb-Smith[2] is polymorphic reticulo-sarcoma. It would seem that in the case here reported the tumour presented in two forms which have received separate classifications. It would serve no useful purpose to theorize in regard to this histological variation, except to suggest that if the biopsy material is accepted as representing an example of a stem cell lymphoma, it could in this instance be said to represent a stage in dedifferentiation of the primary growth, the site of which would seem likely to have been in the mediastinum. One other feature seems worthy of notice in regard to the autopsy material, and that is the presence of abundant histiocytes in association with the neoplastic reticulum cells. It is difficult to know whether to look upon these cells as differentiated derivatives of the reticulum cells and part of the newgrowth in the true sense, or merely as evidence of reaction upon the part of the invaded tissue; certainly they did not differ morphologically from the histiocytes found in the sinuses of

lymph nodes not recognizably neoplastic, or from the histiocytes present in such numbers in the interlobular septa of the lungs.

Conclusion.

A case of reticulo-sarcoma is described with a rapid course, characterized by a persistently raised temperature and respiratory obstruction, and presenting a histological picture which can be divided into two distinct cytological classes.

Acknowledgements.

Our thanks are due to Mr. Woodward-Smith, Department of Medical Artistry, University of Sydney Medical School, for the photomicrographs.

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(1) A. M. Ewing: "Neoplastic Diseases", Fourth Edition, pages 374 and 378.
(2) A. H. T. Robb-Smith: "Reticulosis and Reticulo-Sarcoma; Histological Classification", *The Journal of Pathology and Bacteriology*, Volume XLVII, 1938, page 457.
(3) E. A. Gall and T. B. Mallory: "Malignant Lymphoma; Clinico-Pathological Survey of 618 Cases", *The American Journal of Pathology*, Volume XVIII, 1942, page 381.

Reviews.

CLINICAL SURGERY.

THE seventh edition of "A Short Practice of Surgery", by Hamilton Bailey and R. J. McNeill Love, has just been published.[1] This book, as far as manner of presentation is concerned, is a model for all textbooks. The clearness of the writing, the setting out of the paragraphs, the use of different types of printing, the selection of the many illustrations, make it a very easy book from which to learn. At the same time we must point out what was pointed out in our last review of it—that it is a summary, and that a summary is always harder to learn from than is the complete account. The student will learn most of the facts of surgery from this book, but it cannot be said that he will fully learn the principles. Surgery has become such a large subject that it is no longer possible to put all the necessary knowledge into a manual. Nor is there any great virtue in a manual. Textbooks are not carried about; they are usually read whilst placed on a desk. If a handbook of facts is needed, this book may be used.

"ANCIENT ANODYNES."

A MOST useful contribution to the history of anæsthesia has been made by Dr. E. S. Ellis, late anæsthetist to the Gloucestershire Royal Infirmary, England. While still practising his profession as a specialist anæsthetist, he evidently found time to devote many laborious hours to painstaking research on the historical background of his subject. Fortunately, he has been persuaded to incorporate his copious records in a small book, to which he has given the euphonious title "Ancient Anodynes".[2] No doubt a concomitant interest over the years in the subject of anthropology acted as a further inducement for him to delve deeply into the many strange methods of anæsthesia employed by primitive man and by more sophisticated people belonging to the ancient civilizations. This dual interest may account for the varied bibliography at the end of the book and for all the references being so fully documented.

The many readers who look for light entertainment in their literature may quite conceivably find the earlier chapters rather tiresome, as it is irritating to have subject and epoch juggled about so frequently within the space of a single page. But the innumerable references closely packed into a long series of abbreviated paragraphs have been scrupulously investigated in the true scientific spirit, and must provide a happy hunting-ground for the research student in this department of medical history. The later chapters, on the other hand, are far less disjointed, and when it comes to a discussion on the development of modern anæsthesia, the story is told in a thoroughly consecutive and convincing manner.

In the beginning of the book the author mentions various physical means of relieving a person of sensibility by agents somewhat analogous to the policeman's baton, followed by psychological methods as exemplified by the hypnotic trances under the influence of mesmerism. Next the author gives a number of references to drugs and aromatic substances used in olden times for their soporific effect upon the nerve centres after the process of inhalation. A detailed account is also given of the anæsthetic properties of certain agents under the separate headings of the poppy, alcohol, mandragora, belladonna, *Cannabis indica*, hyoscyamus, hellebore, hemlock, and others less familiar to the medical historian. In a later chapter the same subject is viewed from a different angle with a spate of references about anæsthesia in each country of the ancient world.

Even if this valuable book cannot be conscientiously placed in the category of light reading matter—and there are times when it is capable of exerting a soporific effect all its own—it undoubtedly contains a wealth of reliable information, helpfully documented for the benefit of the research student; and it is doubtful whether any previous work on this particular subject has ever been set out upon exactly similar lines. It is to be hoped that the author will make it the basis for a larger and more harmonious volume in the near future.

MORELL MACKENZIE AND HIS ROYAL PATIENT.

MANY of us have vague recollections of polemical discussions in medical circles which centred around the personality of Sir Morell Mackenzie, the distinguished English throat specialist, and his treatment of a greatly respected member of the German royal family, who came to an untimely end in 1888 from a malignant growth of the larynx. This strange episode in the medical annals of last century has had more than a purely historical interest for Dr. R. Scott Stevenson, a present-day specialist in this branch of medicine: he could not resist the desire to investigate thoroughly the facts of the case in a coldly objective way, so that the controversial elements of the Morell Mackenzie incident might be satisfactorily disposed of once and for all.

The outcome of his research is the small book entitled "Morell Mackenzie", and although it may be still problematical whether the author has completely achieved his purpose, it cannot be denied that he has given a vivid interpretation of that peculiar Victorian tragedy so disastrously connected with the life of Mackenzie; and, coincidently, he reveals in a pleasant style many interesting sidelights on the vagaries of individualistic medical practice and of the intricate European politics of the day.[1] There are also frequent references to a number of new developments in medical science, some of which contributed in no small way to the rise of specialism and to the establishment of special departments at some of the large public hospitals.

At the time of the onset of the fatal illness of the German Crown Prince (who later became the Emperor Frederick III), Sir Morell Mackenzie was considered the outstanding specialist in diseases of the throat, and he had already achieved an international reputation. Furthermore, he was then one of the few medical men in Europe who were expert in the use of the laryngoscope, which had been invented by the singing master, Manuel Garcia, in 1854, and three years later applied to medicine by Turck, of Vienna, and Czermack, of Budapest.

The oto-rhino-laryngologist who is partial to detective stories will be entertained as he reads through these pages in forming his own conclusions as to who was actually the arch villain of the piece. At the same time he may be clever enough to find a clue to the origins of that bitter international rivalry that was to bring in its train so much bloodshed and anguish to succeeding generations.

Without wishing to initiate any further disputatious questions involving the medical profession, it must be claimed in fairness to the eminent Claude Bernard that he, and not the famous Jean Martin Charcot, first used the words "on entering the laboratory, one should divest oneself of the imagination as one does an overcoat".

The book contains a number of apt illustrations, and all the references are carefully documented.

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[1] "A Short Practice of Surgery", by Hamilton Bailey, F.R.C.S. (England), F.I.C.S., and R. J. McNeill Love, M.S. (London), F.R.C.S. (England), F.I.C.S.; Seventh Edition; 1946. London: H. K. Lewis and Company Limited. 8½" × 5½", pp. 1106, with many illustrations, some coloured. Price: 40s. net.

[2] "Ancient Anodynes: Primitive Anæsthesia and Allied Conditions", by E. S. Ellis, M.R.C.S., F.R.A.I., with Foreword by T. K. Penniman, M.A.; 1946. London: William Heinemann (Medical Books) Limited. 8½" × 5½", pp. 192. Price: 21s.

[1] "Morell Mackenzie", by R. Scott Stevenson; 1946. London: William Heinemann (Medical Books) Limited. 8½" × 5½", pp. 202, with illustrations. Price: 15s.

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MEDICAL BENEVOLENCE.

DURING the past few months an effort has been made throughout the Commonwealth to prevail upon members of the medical profession to subscribe to the Federal Medical War Relief Fund, a fund started by the Federal Council of the British Medical Association in Australia for the relief of distress among medical officers of the armed forces in the war of 1939-1945 and their dependants. The first appeal was made by the President of the Federal Council in February, 1946, and in September the treasurer of the council pointed out in a letter to this journal that only 25% of the members of the profession had contributed and that the sum contributed at that time was £15,000, though the original objective was £50,000. This curious lack of generosity is unfortunately shown in the treatment by the medical profession of its general benevolent funds. It has been found necessary every few years to appeal in these columns for support of the last-mentioned funds; the last appeal was made in March, 1940. Important as the Federal Medical War Relief Fund undoubtedly is, medical practitioners should not allow it to divert their attention entirely from the benevolent funds. The benevolent funds were in existence long before special funds to relieve distress caused by the first and second world wars were needed—the Victorian fund was established in 1865 and the New South Wales fund shortly afterwards, in 1868. They would never have come into being if the need for them had not been real. The profession of medicine in Australia has never been impecunious—its members have generally managed to earn a satisfactory living, they have been able to educate their children and to make some kind of provision for their dependants in the event of their own death. But this has not been true of every member. Even in times gone by, when the incomes of many doctors were relatively high and taxation was low, there were always some who, often for no fault of their own, met with misfortune that left them crippled financially, with the result that they and their children suffered, and some, of course, died at an unexpectedly early age before they were able to make any kind of provision. In the days to come this small number will become greater, and for several reasons. There will be more doctors than there were; incomes will not be high; and taxation is not at all likely to return to the pre-war level. We have also to remember that distress is by no means always obvious. Those who administer medical benevolent funds know that acute hardship is often borne in silence and is sometimes discovered only with difficulty.

Some form of medical benevolent fund exists in every State except Queensland. It is curious that Queensland has no fund, for we cannot imagine that there is no need for a fund. In 1941 some discussion arose about the establishment in Queensland of a fund that would serve as a war relief fund and also be applicable for use in civilian circles. The Queensland representatives also pointed out at a meeting of the Federal Council in September, 1942, that the Branch wished to see the establishment of a general relief fund; this idea was not entertained for several reasons. Now that the Federal Medical War Relief Fund has been inaugurated, the Queensland members of the Association may, and it is to be hoped will, think about the establishment of a fund within their own Branch to meet civilian needs. The Victorian Medical Benevolent Association has been active during the eighty-one years of its life. According to the report presented to the annual meeting in November, 1944, the funds of the association amounted to £9,040 and were derived from donations, subscriptions of members and interest on the investment of permanent funds. The committee of the association reported that it had been able during the year to respond to every application for relief. At the same time it viewed with anxiety the fact that in the immediate future additional and more burdensome claims would be made on its funds. When we read that monthly allowances, some of £4, others of £8, are made and that in some instances these allowances have been maintained for several years, we realize that something has been done—the association has "blunted the sharp edge of poverty and brightened the declining years of aged and afflicted members of the profession". At the same time this is not a great deal. More could be done and, as already pointed out, more will need to be done in the future. The association from the point of view of its funds lives a more or less hand-to-mouth existence. This is because a great deal of reliance has to be placed on donations and subscriptions. Thus in 1943 the sum derived from subscriptions was £225; special donations yielded £7 and investments £336. We can well understand the desire of the committee to increase the permanent fund rather than the annual subscriptions. The Medical Benevolent Association of New South Wales comprises as members those who have signed an application form for membership and who have paid a sum of ten guineas in one sum or an annual subscription of one guinea. (Life membership is achieved when a sum of fifteen guineas has been paid in annual subscriptions.) Those who have subscribed without signing an application form for membership are designated subscribers. The association has assets amounting to upwards of £7,500. Last year the sum of £578 15s. 4d. was paid in regular allowances to beneficiaries, and Christmas gifts amounted to £315. The assets of the Medical Benevolent Association

of South Australia, which was established in 1881, amounted at the end of 1945 to upwards of £5,800. Members comprise life members (those who have donated £10 or more in one sum) and subscribers. During 1945 one donation of £25 was received and also three of £10. The sum of £44 7s. was received in subscriptions from 85 doctors. During 1945 the association was able to make some response to all requests for help. In our last reference to this subject in 1940 we reported that the South Australian association expressed disappointment because its financial position was not sound enough to allow it to contribute towards the education of deceased or distressed medical practitioners. Presumably there are no children in need of help at present; if there were the officers of the association would find it difficult to do a great deal with the money at their disposal. The Medical Benevolent Association of Western Australia was founded in January, 1930. It emerged as a separate organization from a subcommittee of the Western Australian Branch of the British Medical Association. It started with seven life members and 81 members. The subscription is one guinea *per annum* and when fifteen guineas have been paid the member becomes a life member; if a single payment of ten guineas is made the member becomes a life member. The fund has never been largely supported, the greatest number of practitioners subscribing being only 97. Since 1930 the sum of £1,062 has been disbursed; in 1945 three beneficiaries received £159. In 1943 the funds were augmented by a special contribution from the War Relief Fund. At the end of 1945 the total accumulated funds amounted to £935. At the end of 1945 most of the subscribers became life members, with the result that only 23 members remained to subscribe one guinea each out of a total of 350 practitioners in the State. The 56 life members agreed to continue to subscribe, but this was insufficient to meet the increased demands on the fund. Following the submission of the annual report of the association to the Western Australian Branch of the British Medical Association the opinion was expressed that most members of the Branch could and should subscribe. It was therefore resolved to increase the annual Branch subscription by the sum of one guinea for the benefit of the fund. The Benevolent Association is thus now assured of an income of between £300 and £400 *per annum*. The fund in Tasmania is quite small and consists of the unexpended portion of an amount that was raised some years ago for a specific purpose.

From this cursory view of the medical benevolent associations in the several States it is quite clear that every one of them could do more if it had more money. Many methods of raising money have been suggested. In many instances what is needed is that the claims of these benevolent funds shall be brought to the notice of members of the Australian Branches at the right moment. We do not believe that doctors deliberately turn a deaf ear to the appeals for help that come from their own ranks. There seems to be a kind of inertia about making a special effort to respond to special appeals. Though in every instance life membership donations are sought rather than annual subscriptions, since they augment the permanent funds and allow budgeting to meet special needs to be undertaken in advance, the expedient has been tried with a certain amount of success of asking members to add to their cheques paid in respect of membership of the British Medical Association Branch an extra sum, with the request that it be handed on to the treasurer of the benevolent fund. Another device was suggested in the report for 1944 of the Victorian Medical Benevolent Association. It was that recognition of services rendered to one another by members of the Association should take the form by mutual agreement of a gift to the permanent benevolent fund in the name of the doctor rendering the service. It appears that this custom has been adopted widely in South Africa and that the result has been the creation of a large fund for benevolent purposes. It may well be considered in this country. Another expedient, mentioned in our discussion of 1940, was suggested in South Australia. It was that members might take out insurance policies for £100 or more, maturing in ten to twenty years, according to the age of the proponent, and assign them to the benevolent association. In addition to all these expedients there is one that should never be forgotten—members of the profession will find it no hardship to bequeath in their wills a small sum to the medical benevolent association of their State; the sum need not be large. If this was done the future of these organizations would be assured.

Throughout this discussion it has been taken for granted that members of the medical profession approve of the existence of associations which can help the needy and that they cherish certain *esprit de corps* for members of their own profession. If they have this feeling they cannot see their brethren have need and shut up "their bowels of compassion" from them. It is after all a case of love of one brother for another. And "let us not love in word, neither in tongue; but in deed and in truth".

Current Comment.

THE INFLUENCE OF BODY TEMPERATURE ON DRUG ACTION.

THE action of drugs on the human subject is known to be modified by a number of factors, of which the most important are age, weight, sex and race, if we regard the last named as corresponding with species as a biological distinction. To these may be added body temperature, though it is probably not often thought to be significant. Probably it is not of great importance in routine drug therapy, since the margin of safety between therapeutic and toxic doses is as a rule ample. However, more active drugs are associated with greater hazards, and it is likely that the future will see more scientific methods used as a basis of drug usage and dosage.

Frederick A. Fuhrman has collected a quantity of interesting and valuable work dealing with the action of drugs in relation to body temperature.[1] The observations he has reviewed have been made from the point of view of the pharmacologist, and naturally are based largely on work on animals of various species. But he points out at the outset that the body temperature of man is capable of variation between wider limits than is generally thought possible, even if we admit that the external circumstances would be unusual. That this consideration is not purely academic may be illustrated by the now familiar modifications accepted in the treatment of shock, following the experiences in this war, in which the unfavourable effects of unwanted heat became evident. A general discussion of the subject must of course include the effect of drugs on animals whose metabolic characteristics are vastly different from those

[1] *Physiological Reviews*, April, 1946.

of man. It might seem futile to compare amphibia and human beings, seeing that a frog and a man at ordinary room temperature may show a difference of body temperature as great as 20° C. But it is found that a drug such as colchicine, whose toxic effects are easily elicited in man, is much more toxic to frogs if their body temperature is raised. Moreover the frog has been used a good deal in the past in assaying digitalis. In this case the influence of season has been found to be definite, and so also has the raising of temperature. When an animal like the cat or dog is used for the same purpose a striking difference is noted in the response to the drug, and such difference may be seen also in isolated organs. There may be altered effects due to species, but there seems to be some reason for believing that some at least of the heightening of response to certain drugs may be due to the metabolic acceleration associated with raising of the body temperature. Those parts of Fuhrman's paper dealing with the purely experimental aspects of physiology cannot be dealt with here, but it is of interest to the clinician to read a collected account of the different behaviour of various drugs on animals under different conditions of body temperature. Such drugs as digitalis and its glucosides, strychnine, "Coramine", caffeine, barbiturates, alcohol, epinephrine, cocaine, and certain of the biological preparations are of great importance in medicine, and accurate knowledge of their effects is desirable. Fuhrman points out, however, that it is not possible to say dogmatically that a simple raising of the temperature of the body will enhance a drug's effects, a proposition put forward by Richet many years ago. It is more helpful to distinguish between the variations in the rate at which drugs will permeate the tissues at raised temperatures, and variations in the concentrations and mode of action of such substances. One most important factor to be remembered in the practice of medicine is the effect of body temperature on circulation time. Thus the optimum dose may be conditioned by the optimum time of administration if attention is paid to temperature as well as other considerations. Medical officers who have of recent years treated men with relapsing fever will recall that it is usually thought advisable to defer the intravenous administration of arsenic until the acuteness of a rigor is over.

One interesting point raised by the author is that of detoxication of drugs in the body. The method by which this is accomplished is not always known, but it is believed that in the case of some drugs, like atropine, epinephrine and alcohol, and perhaps also the barbiturates, an enzymatic action is at work. Temperature would be of obvious significance here, and the persistence of the action of some drugs during hypothermia is well recognized. Fuhrman concludes his article with a quotation from Brunton, who will be remembered by the more senior as an authority in his day: "All these things show that the definition of the action of a drug must be still further modified, and we must define it as the reaction between the drug and the various parts of the body at a certain temperature."

THE INFLUENCE OF USE AND DISUSE IN THE REPAIR OF NERVES.

THE introduction of sulphonamides and penicillin and more refined knowledge of the action of antiseptics have undoubtedly stimulated reparative surgery and encouraged the surgeon to take the bolder and earlier action that his desires have urged. This again has stimulated renewed interest in the restoration of interruptions in the neuromuscular chain, which is so essential for our proper activities. The physiologist's field here, as elsewhere, touches that of the practising doctor. J. Z. Young, in his Sydney Ringer Memorial Lecture, links structure and function as an anatomist should, and has shown how some of the gaps are being filled in our knowledge of the relations of nerve and muscle.[1] He points out how the phenomena of atrophy and hypertrophy in response to disuse or maintained use are still in some ways obscure, and is inclined to doubt the validity of the conventional view that heredity and function are in their influence more or less distinct. Both forces may act early or late, for reproduction within the body is a process which goes on and on, and structure and function must be considered together. Thus in the study of Wallerian degeneration it is not enough to regard the atrophy of a muscle cut off from its cell as resulting solely from disuse. It is due also to the breaking up of the integrity of a balanced system. Nerve cells range in size from the large to the small, and this size is related to individual function, the motor fibres being larger than the cutaneous. When a nerve regenerates after division, it is obvious that the growing fibres will not of necessity occupy the same neurolemmal channels, yet after a year of regenerative activity the motor bundles contain larger bundles than the cutaneous as before. Evidently it is something more than the influence of the channel down which the fibres have grown that has dictated their size. But experiment has shown, as Young emphasizes, that if a nerve is severed functionally without destruction of the fibre channels, with the production of what is now called axonotmesis, large fibres will form only if they are permitted to make connexion with their proper peripheral destination. Further, hypertrophy and atrophy, as illustrated in a nerve growing under such conditions, are found to be conditioned not only by the central but also by the peripheral connexions of the nerve. Young refers also to the problem which has attracted neuropathologists, the atrophy of nerve cells when deprived of their fibres, and the related problem of the behaviour of peripheral tissues when deprived of central control. It is interesting that denervated tissues show hyperexcitability to certain mechanical and chemical stimulants, for example, in appropriate cases, acetylcholine.

It is natural that the histologist should be curious about the details of the method by which nerves and muscles establish their ultimate connexions. There is apparently a sharp boundary between the two, yet denervated muscle, as we know, undergoes rapid atrophic changes, both physical and chemical. Such changes are not confined to muscles deprived of nervous connexions, but occur also in muscles deprived of the opportunity to resist counter-action of other muscles. Splinted muscles may at least be partly saved from atrophy by keeping them judiciously stretched, by encouraging daily voluntary contraction and by galvanism. In the last-named method the work of J. C. Eccles is quoted, which suggests that if a denervated muscle is allowed to shorten during electrical stimulation the degree of wasting is lessened. Young concludes by enunciating the principle of double dependence. This maintains that the existence of each component of the neuro-muscular system depends not only on blood supply and nutrition, but also on the action of a stimulating or constraining influence from above and the ability of this higher authority to produce stimulation or constraint below. Hypertrophy can take place only when the tissues can act so as to assist the life of the body; remove this teleological function and atrophy will be the result. The application of these principles is familiar in treatment. But when an important nerve is severed, what is to be done? War injuries have made the answer to this question more urgent. The rate of nerve regeneration can be calculated in any given case, but where a long distance has to be traversed a long wait is necessary before it can be determined if Nature can tackle the job unaided or whether surgical help will be required. Meanwhile, not only time but precious muscle function may be lost. This dilemma of orthopædic and neural surgeons should now be less productive of anxiety. On the one hand the physiological principles are clearer, for surgical thought and therapy in any individual case range over the whole length of the neuronic path affected. On the other hand, bolder action is now encouraged by the greater safety of the procedures of exploration and repair. In this, as in other branches of reparative surgery, the advances are not only in technical procedures but also in the attitude and equipment of the mind that directs the treatment.

[1] *The Lancet*, July 27, 1946.

Abstracts from Medical Literature.

DERMATOLOGY.

Clinical Investigation of a New Entity.

C. L. SCHMITT, O. ALPINS AND G. CHAMBERS (*Archives of Dermatology and Syphilology*, October, 1945) call attention to an unusual eruption which occurred amongst the military personnel in the South-West Pacific area. The affected persons were members of the Australian and American forces whose stay in the tropics ranged from two to sixteen months; the first being seen in October, 1943. The syndrome invariably began with an initial eruption which varied widely in different persons. This early eruption mimicked such common dermatoses as heat rash, fungous infection, eczema, urticaria and contact dermatitis. After the initial picture there followed characteristic firm violaceous elevated papules, nodules or plaques, which, in some cases, remained mild and localized, while in others the involvement was severe and widespread and at times was accompanied by a potentially fatal acute exfoliative dermatitis. Not until the same syndrome was repeated in an increasing number of patients did the authors realize that they were dealing with a new entity. They were unable to find any reports of a similar syndrome in the literature. From two to eight weeks after the onset of the primary eruption there appeared bilateral and roughly symmetrical elevated, firm, rounded or elongated, violaceous papules and nodules which ranged in size from 0·8 to 4·0 centimetres. At first these lesions were soft and smooth-surfaced; later they became leathery and verrucose. The large papules began as such and were never the result of coalescence of small papules. Widely separated groups or patches of violaceous papules with an occasional verrucose nodule comprised the entire picture in the patients with milder eruptions. In addition to papules many nodules and plateau-like elevated patches with some violaceous colour and leathery surfaces also occurred. Deeply violaceous pea-sized sessile warty excrescences, having a predilection for the inner surfaces of the thighs and the suprapubic regions, were present in the patients with more serious involvement. The covered parts of the body were involved more frequently than the exposed areas. The waistline, suprapubic region, intergluteal fold, hands, eyelids, groins, axillary folds, helixes of the ears, shaft of the penis, scrotum, arms, feet, legs, chest and back were involved in frequency about in the order named. Approximately one-third of the patients presented discrete and confluent, tense, greyish-white papules on the lower lip and reticulated, streaked, firm greyish-white patches on the buccal mucous membrane and the lateral and dorsal surfaces of the tongue. Four patients had elevated, irregular, ulcerated, hypertrophic tumefactions on the buccal mucosa and lateral margins of the tongue. Careful search revealed no papules on the *glans penis* in any case. The mucosa of the anus was involved by scaly, slightly infiltrated plaques in four cases. No subjective symptoms were associated with the beginning of the eruption. As the primary lesions progressed, moderate to severe pruritus was common. The itching gradually diminished after the elevated secondary lesions were well established. Constitutional symptoms occurred only with widespread erythroderma and exfoliative dermatitis. The inguinal lymph nodes were often enlarged and tender. During the authors' observations and study, over a period of seven months, changes were still occurring in the patients. The following sequelæ were noted: (*a*) pigmentary dystrophy, (*b*) cutaneous reticulated atrophy occurring at the site of the papules in severe eruptions, the atrophy on the hands being confluent, (*c*) alopecia, (*d*) involvement of nails, (*e*) disturbances of the sweat glands and sebaceous glands. Laboratory investigation failed to reveal anything abnormal. The only observation of significance was the presence of malarial parasites in about one-third of the patients. In regard to ætiology after many possible agents and elements were carefully considered, there remained only one constant factor and that was quinacrine hydrochloride. The authors deduced that it was possible that the drug, either alone or in combination with other factors, produced the peculiar eruption.

Pityriasis Rubra Pilaris and Vitamin A.

ARTHUR D. PORTER AND E. W. GODDING (*The British Journal of Dermatology and Syphilis*, November-December, 1945) state that a difference of opinion exists as to the value of vitamin *A* in the treatment of *pityriasis rubra pilaris*. They quote a case of a woman, aged forty years, first seen in 1942. She had then had skin trouble for three months only. The appearance was that of a severe case of *pityriasis rubra pilaris*. Briefly, her face, body and limbs were covered with a scaly red eruption resembling widespread psoriasis, especially on the palms and soles. The appearance of the scalp closely simulated that of seborrhœic dermatitis. Characteristic follicular keratotic plugs were scattered profusely over the shoulders, neck and thighs, and to a lesser degree elsewhere. A dark adaptation test (as described by Yudkin, 1921) carried out at the same time showed that the patient's power of adaptation was subnormal, the rate being significantly slow for the whole range of adaptation tested. On December 1, 1942, vitamin *A* therapy was begun and continued without intermission for 38 weeks. The daily dose was 33,000 units, and altogether 8,778,000 units of vitamin *A* were given. During this time repeated dark adaptation tests were carried out, the final tests under treatment showing a significant improvement in the rate of adaptation. The only other treatment given was calamine lotion and *Mistura alba*, neither of which, it is believed, could have had any marked effect on the skin. During the first six months of the administration of vitamin *A*, great improvement in the condition of the skin was noted. The redness and scaliness decreased, and the skin became soft and of normal colour. Follicular plugging was no longer apparent, except in certain areas of the thighs. The psoriasiform lesions on the elbows, palms and soles gradually faded away until they were scarcely visible. The scalp, however, still showed the appearance of seborrhœic dermatitis, though it was now very mild. The improvement continued during the following months, but more slowly. The follicular keratotic lesions on the thighs gradually became less and less marked until ultimately they could not be detected. The skin of the palms and soles became normal. The only visible abnormality remaining was the appearance of very mild seborrhœic dermatitis of the scalp, forehead and left ear. Vitamin *A* supplements to the diet have now been withheld for over four months, in an attempt to find out whether the improvement would be maintained. So far there has been no regression in the condition of the skin. The dark adaptation curve, however, suggests that any vitamin *A* stored is almost used up, so it is possible that the condition of the skin will soon deteriorate. It is estimated that the approximate vitamin *A* intake from the diet is not less than 1,500 units per day. In attributing improvement to vitamin *A*, the authors realize that it is unwise to draw conclusions from a single case, especially in a disease which is liable to spontaneous remissions, and which has occasionally been reported to respond to other measures, such as endocrine therapy. While it cannot be maintained that *pityriasis rubra pilaris* is purely a deficiency disease, yet the clinical and histological appearance of the skin, the therapeutic effect of big doses of vitamin *A* and the dark adaptation tests do indicate that vitamin *A* is implicated at some stage. The ætiology of the disease in regard to vitamin *A* remains at the moment largely speculative. Whatever the cause, it appears that liability to the disease is inherited. The records are too few and too lacking in essential details to allow the nature of the inheritance to be stated with certainty, but it seems to be that of a dominant, the dominancy being incomplete or prevented from developing by other genes in certain cases (Cockayne, 1933).

The Treatment of Seborrhœic Dermatitis.

R. M. BOLAM (*The Practitioner*, May, 1946) lists three types of eruption included under the heading of seborrhœic dermatitis. The first type comprises acute spreading lesions of erythematous type on the face and neck, associated with a pronounced degree of seborrhœa of the scalp. The second type is characterized by an extensive acute spread of small oval or circular erythematosquamous patches of dermatitis on trunk and limbs. Thirdly, the author mentions the acute spreading infective seborrhœic dermatitis, involving scalp, ears, axillæ and groin in particular. He states that this last is one of the most difficult skin conditions to treat successfully. It usually supervenes on a severe *seborrhœa capitis* with retroauricular fissuring and an erythematosquamous rash in the axillæ, on the chest and in the pubic region. Investigation shows that a careful history should be taken and a search made for any factor which has contributed materially to the onset of the acute infective spread. Special attention should be paid to the psychological background; to the presence of any disease in the nose, accessory sinuses or middle ear; to diet; to the use of unsuitable methods in the application of treatment which has already been

tried; to the use of unsuitable preparations as a precipitating factor in the present outbreak; to the culture of the organisms from infected sites. In regard to treatment, rest, both physical and mental, and efficient nursing are emphasized. With regard to diet, it is generally agreed that the seborrhœic patient should curtail the intake of carbohydrates, starches and fried foods, and increase the amount of protein, fruit and fresh vegetables with some, at least, of the latter taken in a raw state. Diet supplements are as follows: fat-soluble vitamins *A* and *D* in the form of cod liver oil, halibut liver oil or "Adexolin"; vitamin *C* in the form of ascorbic acid, 150 milligrammes daily; vitamin *B* complex is present in brewer's yeast, baker's yeast and "Bemax", which are recommended in this condition. Internal medication includes, as well as sedatives and alkalis, iron and arsenic and penicillin. With regard to iron and arsenic, many of the patients with chronic seborrhœic dermatitis show definite hypochromic anæmia on investigation and the taking of full doses of iron and ammonium citrate, thirty grains three times a day, with arsenical solution, five minims, in a mixture, is indicated, and must be continued for several weeks to bring about the desired improvement. The use of penicillin may also be justifiable in the cases of heavy infection, and it may be given intramuscularly if the predominant organisms in a culture are sensitive to this drug. The total dose is an arbitrary figure which must be dependent upon the improvement shown in each individual. For local treatment the author mentions baths, bran and alkaline. For the former, a muslin bag containing three pounds of bran is tied firmly at the neck and held beneath the tap while the hot water is run; twenty-five gallons of water at a temperature of 98° F. constitutes the bath. For the alkaline bath, sodium carbonate or potassium carbonate, two ounces, is added to the bath. The author also gives local treatment for the scalp, the ears, and the body and limbs. For the scalp in an acutely inflamed state a saline compress or starch poultice is often the only remedy that can be tolerated. Liquid penicillin (250 units per millilitre) may be sprayed onto the scalp with success in some cases, or penicillin cream of 500 units per gramme may be used. These remedies have their use in the treatment of impetigo lesions on the face and ears. A weak mercury, sulphur and salicylic pomade with an oily base should be applied gently to counteract the dryness and scaling, and after a time careful shampooing with liquid soap may be included in the scalp toilet. The ears always present an awkward problem in treatment. Septic material collects in the deep fissure behind the ear, and adverse conditions of heat and movement prevail when the head comes in contact with the pillow in bed. Preliminary cleansing to remove crusts or discharge should be carried out, and this may be followed by the application of penicillin spray or cream, as used for the scalp, if bacteriological examination indicates that this form of treatment is likely to be successful. More often, however, soaks have to be relied upon. Burow's solution may lessen the secretion and dry the skin over the back of the ear and adjacent scalp. Following this, a 1% ichthyol paste is applied and the part is cleaned gently with liquid paraffin as necessary. For the body and limbs in the acute phase with exudation the simplest of remedies should be employed, and a saline soak is recommended. After two days of dressings repeated every four hours, soaks may be replaced by zinc oxide and starch in a water-miscible base. Preparations containing sulphur and tar are best reserved for the later stage of quiescence and recovery, and even then should be used in low percentage strength, or a flare-up may ensue. A weak ichthyol and zinc cream applied twice daily to the body and limbs is often effective.

Penicillin.

C. HURIEZ AND J. LEBORGUE (*La presse médicale*, March 9, 1946) discuss the treatment of cutaneous infections with penicillin. Staphylococcal infections of the skin from simple folliculitis to septicæmia responded well; *sycosis barbæ* and anthrax and severe impetigo were also successfully treated. Penicillin powder, 500 units per millilitre, or ointment of a strength of 250 to 500 units per gramme of lano-"Vaseline", was used. In resistant cases intramuscular injections of penicillin were used in combination with local treatment. Anthrax was treated with instillation at the base of the lesion of ten millilitres of penicillin (4,000 units per millilitre) every twelve hours or once every twenty-four hours. Streptococcal lesions usually responded to sulphadiazine, but when resistant, penicillin was often successful. Erysipelas also responded satisfactorily. Exfoliative dermatitis and bullous and pemphigus lesions sometimes responded to penicillin.

UROLOGY.

Repair of Hypospadias.

C. D. GOODHOPE (*The Journal of Urology*, November, 1945) states that the literature contains many differing techniques for construction of the penile portion of the urethra in hypospadias, an indication that no one technique is satisfactory in all or even in most cases. He considers that a simplification in the method of using stainless steel wire may help in the matter. Stainless steel wire causes much less tissue reaction than conventional suture material, but it is difficult to handle. The author proposes a method of using it which saves time, and therefore saves much handling with consequent trauma to the delicate tissues involved. An eight-inch length of number 30 gauge wire is wedged into a four-and-a-half-inch straight atraumatic needle. The type of operation is essentially that described by Reed Nesbit. About nine to twelve months after the preliminary stage of straightening the penis, by removal of all vestiges of the fibrosed *corpus spongiosum* and urethra, with filling in of the gap by skin swung over from the apron-like prepuce, construction of a new urethra is embarked upon. The new canal is closed by means of a continuous sliding wire suture, which takes a little bite in each side alternately by means of alternate placing of the point of the long, straight needle. When the needle is pulled through, the wire is kept in place by means of a split lead shot at each end. The bare area underneath and at the sides of the newly made canal is covered up by swinging up a split tube graft, which has been constructed in the first stage from the skin of the scrotum. The lateral edges of this covering layer are sutured by wire in a similar way. Suprapubic cystostomy deviates the urine.

Retropubic Prostatectomy.

T. MILLIN (*The British Journal of Urology*, March, 1946) declares that his new operation of extravesical or retropubic prostatectomy is the most universally applicable operation yet found to eliminate the evils of the obstructing prostate. After an experience of 85 cases it gave him better results than any method he had tried, and this was true of every type of case except the fibroses. The postoperative course was easier than after the Harris operation and far easier than after the Freyer type. The Harris operation, however, was sometimes complicated by post-operative obstruction and urinary incontinence. The retropubic operation was virtually a type of perineal operation performed well away from the rectum. The normal anatomy of the approach presented no important structures which needed to be sacrified. Hæmorrhage was usually negligible. Only a short mid-line incision was necessary. The incision into the anterior aspect of the prostate was of an inverted V type opening up as a lozenge shape. After the enucleation of the adenoma from within the sheath formed by the compressed true prostate, drainage was maintained by a Harris catheter passed through the urethra into the bladder, and this was removed at the end of a week or less. There were four deaths in 85 cases, two from pyelonephritis and two from cardiac failure. The operation was suitable for all types of innocent prostatic obstruction, except the fibroses in which endoscopic resection should be performed.

Trichomonas Vaginitis.

J. NOVAK (*The Urologic and Cutaneous Review*, February, 1946) describes a simple method of cure of vaginitis due to the *Trichomonas vaginalis*. Trichomonas urethritis sometimes complicates the picture, and it can occur in the male as well as in the female. The important thing is to cure the vaginitis and the female urethritis will get better spontaneously. The treatment consists of vaginal douches of 0·5% zinc chloride solution which are used twice a day during the first week and once a day during the second. Fifty grammes of zinc chloride are dissolved in 150 millilitres of distilled water; one tablespoon by measure of this solution is put into a quart of lukewarm water for the vaginal douche. The physician may carry out an occasional thorough vaginal swabbing himself. Sexual relations are strictly forbidden during treatment, and relapses afterwards are usually due to cohabitation with a male trichomonas carrier. The patient's complaints usually vanish within two days, and the trichomonas itself disappears in a few days. The vaginal secretion becomes very scant, whitish and cheesy, and contains only flat epithelial cells with small nuclei. The practical efficiency of the treatment is surprising, since laboratory experiments have classed zinc chloride among the least efficient trichomonacides. Apparently its efficiency results from its action on the vaginal mucosa.

Bibliography of Scientific and Industrial Reports.[1]

THE RESULTS OF WAR-TIME RESEARCH.

During the war a great deal of research was carried out under the auspices of the Allied Governments. It has been decided to release for general use a large proportion of the results of this research, together with information taken from former enemy countries as a form of reparations. With this end in view, the United States Department of Commerce, through its Publication Board, is making a weekly issue of abstracts of reports in the form of a "Bibliography of Scientific and Industrial Reports". This bibliography is now being received in Australia, and relevant extracts are reproduced hereunder.

Copies of the original reports may be obtained in two ways: (a) Microfilm or photostat copies may be purchased from the United States through the Council for Scientific and Industrial Research Information Service. Those desiring to avail themselves of this service should send the Australian equivalent of the net quoted United States price to the Council for Scientific and Industrial Research Information Service, 425, St. Kilda Road, Melbourne, S.C.2, and quote the PB number, author's name, and the subject of the abstract. All other charges will be borne by the Council for Scientific and Industrial Research. (b) Those marked with an asterisk may be obtained by approved applicants without cost on making application to the Secondary Industries Division of the Ministry of Post-War Reconstruction, Wentworth House, 203, Collins Street, Melbourne, C.1. Copies of these are available for reference in public libraries.

Further information on subjects covered in the reports and kindred subjects may be obtained by approaching the Council for Scientific and Industrial Research Information Service, the Secondary Industries Division of the Ministry of Post-War Reconstruction, or the Munitions Supply Laboratories (Technical Information Section), Maribyrnong, Victoria.

CAFT GROUP 6. Dr. Meeson, Brain Research Institute, Neustadt, Baden. (BIOS Evaluation Report 203.) Off. Pub. Bd., Report, PB 11362. 1945. 2 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report contains a statement by Dr. Meeson on his collaboration with Professor Vogt on research of the effects of carbon dioxide on the brain.

GERMANY. REICHSFORSCHUNGSRAT. *Arbeitsgemeinschaft für Seuchenforschung.* Gen. Arzt. Prof. Dr. Schreiber. (Lectures and correspondence dealing with diseases occurring in epidemics, especially contagious hepatitis.) (RFR 277.) Off. Pub. Bd., Report, PB 12866. 1943-1944. 258 pp. Price: Microfilm, $3.00; Photostat, $18.00.

GERMANY. REICHSFORSHUNGSRAT. *Der Leiter der Fachsparte allgemeine (klassische) Medizin.* (General correspondence and reports on medical research projects.) (RFR 274.) Off. Pub. Bd., Report, PB 12863. 1943-1944. 76 pp. Price: Microfilm, $1.00; Photostat, $6.00.

GERMANY. REICHSFORSCHUNGSRAT. Prasidialratsmitglied Gen-Arzt Prof. Brandt. (Reports on synthetic substitutes for catgut used in surgery.) (RFR 208.) Off. Pub. Bd., Report, PB 12762. 1943-1944. 56 pp. Price: Microfilm, $1.00; Photostat, $4.00.

GERMANY. REICHSFORSCHUNGSRAT. Prof. Achelis. (Physiological research by Prof. Achelis on skin sensitivity.) (RFR 2.) Off. Pub. Bd., Report, PB 13169. 1943-1944. 9 pp. Price: Microfilm, 50c.; Photostat, $1.00.

GERMANY. REICHSFORSCHUNGSRAT. *Reichsinstitut zur Erforschung und Verhütung der Staublungenerkrankungen.* (Plans for research and prevention of lung diseases caused by dust in industry (pneumoconiosis).) (RFR 261.) Off. Pub. Bd., Report, PB 12851. 1943-1944. 104 pp. Price: Microfilm, $1.50; Photostat, $7.00.

MUSSEMEIER. *Weitere Untersuchungen über den sogenannten Herztod der Schweine.* (Further investigations into the so-called cardiac death of the swine.) Off. Pub. Bd., Report, PB 13204. 1943. 20 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The cardiac death of the swine is shown to be closely related to a hyperfunction or dysfunction of the thyreoid gland (hyperthyreoidism or thyreotoxicosis). Feeding tests have shown that these diseases are the result of one-sided nutrition, especially of foodstuffs rich in potassium and deficient in sodium and calcium. Such a condition can be brought about by feeding too much potatoes and fishmeal and too little green fodder and skimmed milk. The prevention and therapy, therefore, consist in a well-balanced diet and, in addition, in the administration of vitamin *A*, some fluorine compounds (mono-fluor-thyrosin), and salts, especially NaCl, Na_2SO_4, $CaCO_3$, MgO, $MgSO_4$, and calcium lactate. Removed from ALSOS *Reichsforschungerat* File 244.

WALTON, D. C. The testing of chemical warfare agents by the surviving tissue method. (Chemical Warfare Service, Edgewood Arsenal Technical Report, E.A.T.R. 41.) Off. Pub. Bd., Report, PB 6256. 1930. 18 pp. Price: Microfilm, 50c.; Photostat, 2.00.

Report of tests made at Edgewood Arsenal, 1928 to 1930, to determine if the use of segments of rabbit intestines in physiological solutions would provide a suitable method for the comparison of the physiological action of different toxic or irritant arsenicals and for testing the therapeutic value of suggested cures for poisoning from arsenicals. It was found that the method is too complex and laborious for ordinary use and not practicable as a substitute for animal experiments.

ALEXANDER, S. F., GINSBURG, T. H., AND MICHAEL, H. O. The use of concentrated plasma in conjunction with oxygen as the treatment of phosgene poisoning in the rabbit. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 43.) Off. Pub. Bd., Report, PB 13664. 1942. 6 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This report is a sequel to MD(EA) Memorandum Report 34 (PB 13672), by R. F. Sledge and others, entitled: The use of bovine albumin solution, normal rabbit plasma and concentrated rabbit plasma in the treatment of phosgene poisoning, in which the use of plasma for the treatment of phosgene poisoning in rabbits was found to be of no value. In the present report, concentrated plasma, used in conjunction with the inhalation of oxygen, was likewise ineffective and seems to have had a definitely deleterious effect. A table is included.

BOWERS, RUSSELL V., GINSBURG, THEODORE H., AND SHILS, MAURICE E. The effect of inhalation and intratracheal injection of demulsifying agents on the frothy pulmonary exudate in rabbits following exposure to phosgene. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 79.) Off. Pub. Bd., Report, PB 11346. 1943. 8 pp. Price: Microfilm, 50c.; Photostat, $1.00.

When rabbits were gassed with phosgene and subjected to sprays of water solution of agents such as acetic acid, tannic acid, "Foam Killer", caprylic alcohol and "Papain", there followed no consistent or significant alteration in the death rate of animals or in the character or amount of the pulmonary exudate. The death rate of rabbits treated with intratracheal injections of water solutions of "Foam Killer" and calcium chloride was higher than that of the untreated controls. Tables and a bibliography are included.

DAMON, ALBERT. Anthropological survey of Army Air Force cadets. (Army Air Forces. Experimental Engineering Section, EXP-M-49-695-4A.) Off. Pub. Bd., Report, PB 6937. 1942. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Conferences were held with Dr. E. A. Hooton, Anthropology Department, and with Dr. S. S. Stevens, Psycho-Acoustic Laboratory, Harvard University. The basic analysis of the anthropological survey by Harvard University is now complete. This comprises frequency and percentile distributions, averages and ranges of some fifty body measurements and qualitative observations on 2,901 aviation cadets and 584 gunners. The body size survey and the three subsequent facial surveys of aviation cadets, Negro R.O.T.C. cadets, and flying officers and enlisted men, provide a rich store of information for any purpose relating to physique in Army Air Force flying personnel. Further studies are suggested. Appended is a report by Albert Damon on size and location of the external ear based on anthropological surveys by the Aero Medical Research Laboratory which tabulates data obtained.

HICKAM, JOHN B. The wounding effect of 90 mm. H.E. shell fragments. (Army Air Forces. Engineering Division, ENG-49-697-7.) Off. Pub. Bd., Report, PB 5170. 1944. 28 pp. Price: Microfilm, 50c.; Photostat, $2.00.

This is a report of experiments made by the Aero Medical Laboratory to investigate the wounds made by a 90 millimetre H.E. shell at close range to the burst. Sheep were used as experimental animals. In general, they were anæsthetized with "Nembutal" and laid on the left side in wooden frames. They were placed to receive fragments passing at a 50° to 60° angle from the projectiles' line of flight, about 35 feet from the burst. The report describes the characteristics of wounds made by high-velocity casing fragments, extension of damage from the fragment track, wounding power of small, high-velocity fragments, and the effect of the flak suit on high-velocity fragment wounds. References are included.

KOONTZ, A. R., AND MOULTON, C. H. "Avertin" and "Salyrgan" in the treatment of lung œdema induced by chemical irritation. (Chemical Warfare Service. Edgewood Arsenal Technical

[1] Supplied by the Information Service of the Council for Scientific and Industrial Research.

Report, E.A.T.R. 185.) Off. Pub. Bd., Report, PB 6320. 1934. 20 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The work reported here was a continuation of the experiments reported in E.A.T.R. 65. The present report deals with the treatment of œdema of the lungs (induced by chemical irritation) by "Avertin" narcosis supplemented by intramuscular injections of "Salyrgan". It was concluded that the use of "Avertin" *per rectum* not only reduces the death rate following gassing with phosgene (E.A.T.R. 65), but also limits the development of œdema of the lungs (as measured by the lung-heart index). The use of "Salyrgan" intramuscularly, administered independently or as an adjuvant to "Avertin" narcosis, was of no value in reducing the death rate or in limiting the œdema of the lungs following gas with phosgene.

LAUGHLIN, ROBERT C. The effectiveness of irrigation of the human eye using the army canteen. (Chemical Warfare Service. Medical Research Laboratory MRL(EA) Report 34.) Off. Pub. Bd., Report, PB 9560. 1944. 16 pp. Price: Microfilm, 50c.; Photostat, $2.00.

A solution of homatropine in olive oil was prepared in such strength that one drop would cause submaximal dilatation of the pupil. One drop of the solution was placed in each eye of 57 human volunteers who were treated as follows: Group 1: controls, no treatment. Group 2: one eye irrigated by investigator with 60 c.cm. water using rubber bulb syringe. Group 3: one eye irrigated by another soldier using 450 c.cm. water from standard canteen. Group 4: one eye irrigated by another soldier using modified canteen. Group 5: self-irrigation of one eye using standard canteen. Group 6: self-irrigation of one eye with modified canteen. Irrigation by the soldier using the canteen was not as effective as irrigation by the rubber bulb syringe. Tables present test data.

LINTHICUM, E. S. Value of protective ointments against mustard vapour and recommendations concerning future work. (Chemical Warfare Service. Edgewood Arsenal Technical Report, E.A.T.R. 25.) Off. Pub. Bd., Report, PB 6288. 1930. 25 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The object of this report was to review the literature on the value of protective ointments against mustard with a view to recommending whether or not further work should be done at this time. A critical review has been made of all former work on the subject of protective pastes, and the experience gained by actual use in the field has been evaluated. It was concluded that, although some type of protection is needed for the individual soldier against the effect of vesicants, the prospect of definite accomplishment in the direction of a protective ointment is low. It was recommended that work on the project of a protective ointment be delayed indefinitely.

MACCARDLE, ROSS C. Effects of rapid decompression on animals. (Army Air Forces. Experimental Engineering Section. EXP-M-49-696-22.) Off. Pub. Bd., Report, PB 5155. 1942. 24 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The purpose of this memorandum is to submit a preliminary report on some histopathological and physiological effects of rapid and explosive decompression on normal and on choline-deficient animals. The effect on normal animals of explosive decompression from ground level or from 10,000 feet to an altitude of 50,000 feet is summarized in Appendix I, the experimental technique is described in detail in Appendix II, and records of experimental animals at simulated high altitudes in low-pressure chambers are given in Appendix III. Table 1 summarizes the experimental use of the animals; Table 2 summarizes the gross pathologies found in animals subjected to one or more decompressions during the course of an experiment; Table 3 gives summary of experiments on chronically exposed animals; and Table 4 shows blood pressure responses to rapid decompression. Six figures also included.

PENROD, KENNETH E. Studies of respiratory ventilation of fighter pilots. (Army Air Forces. Experimental Engineering Section, EXP-M-49-696-25.) Off. Pub. Bd., Report, PB 5156. 1942. 11 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The purpose of this memorandum is to report measurement of respiratory ventilation of fighter pilots in different airplanes at different altitudes and in various states of activity. Flight tests were made to determine the amount of oxygen required for a fighter aircraft. In order to accomplish this, measurements by methods described in Appendix 1 were made of respiratory minute volume under a variety of conditions. Data on flying personnel may be found in Appendix 2. Individual variation in lung ventilation at 10,000 feet altitude is tabulated in Appendix 3. Effect of altitude on lung ventilation is tabulated in Appendix 4. Comparison of lung ventilation while flying four types of airplanes at 10,000 feet is tabulated in Appendix 5. The effect of combat on lung ventilation is tabulated in Appendix 6, and comparison of lung ventilation observed in operating a link trainer in smooth weather and flying a fighter airplane straight and level at 10,000 feet is tabulated in Appendix 7.

RENSHAW, BIRDSEY. Summary report on "Miscellaneous toxicological studies", to September 1, 1945. (OSRD Report 5669.) Off. Pub. Bd., Report, PB 5927. 1945. 15 pp. Price: Microfilm, 50c.; Photostat, $1.00.

This is the draft of one of the chapters being prepared by the Rockefeller Institute for Medical Research for the NDRC Division 9, Summary Technical Report. Four investigations summarized are: (i) the pathological changes caused by prolonged exposures to oil screening smokes, (ii) the toxic effects of gasoline fumes, (iii) the toxicity of Salcomine dusts, and (iv) the hypersensitivity and dermatitis caused by hexanitrodiphenylamine and enemy explosives containing it.

SALZBERG, P. L., *et alii*. Protective and therapeutic agents for war gases—preparation of new antidotes, to June 15, 1945. (OSRD Report 4460.) Off. Pub. Bd., Report, PB 5952. 1944. 53 pp. Price: Microfilm, $1.00; Photostat, $4.00.

Because of the success of BAL as an antidote for Lewisite, it was hoped to develop a similar antidote for mustard. The greater part of the synthesis programme was directed to the preparation of thiols or compounds potentially capable of developing thiols by elementary processes of rearrangement, hydrolysis, or biological assimilation. Of over 100 compounds submitted, none showed promise in treatment of liquid H skin burns. There was some evidence that the dithiocarbamates and o-aminothiophenol series lessen the effect of H in the eye. Tables are included. Thirty-nine references. This is a progress report under contract with E. I. du Pont de Nemours and Company.

SMITH, HOMER W., *et alii*. The comparative systemic effects of mustard and the nitrogen mustards (HN1, HN2, HN3) in rats, to May 1, 1945. (OSRD Report 5180.) Off. Pub. Bd., Report, PB 5875. 1945. 20 pp. Price: Microfilm, 50c.; Photostat, $2.00.

After gassing and percutaneous administration, the systemic effects of the nitrogen mustards in albino rats at dosage in the range of the LD_{50} or LC_{50}, and about 0·5 LD_{50} or LC_{50}, were more severe than after H, with the single exception that HN1 was less myelotoxic than the other four compounds. On the basis of dosage required, HN_3 was the most efficient in producing these effects. Intravenous and subcutaneous injection of H in propylene glycol reverses the relationship; at the LD_{50} dosage it was most consistent and most toxic judging by the loss of weight, leucopenia, extent of lymphoid atrophy, bone marrow destruction and enteritis. HN_2 was again the most efficient of the nitrogen mustards and was approximately equivalent to H. HN1 at the dosage employed was least leucotoxic. Tables, charts and bibliography are included. This is a progress report under contract with the New York University. Experimental work was done by Irving Graef, Val B. Jager and David Karnofsky.

SMITH, HOMER W. Progress report on review of the literature on the systemic action of mustard gas to August 1, 1943. (OSRD Report 1717.) Off. Pub. Bd., Report, PB 6013. 1943. 58 pp. Price: Microfilm, $1.00; Photostat, $4.00.

This report reviews the available literature on the evidences of systemic intoxication produced by mustard in experimental animals and in man. In preparing this review the author has had the advantages of the recent studies of his associates and of other investigators on the action of the nitrogen mustards in experimental animals, as well as a limited experience with mustard itself. His selection of odds and ends from clinical notes and his occasional interpretive bias have been influenced by this fact. All discussion of the pathology of the eyes, skin and respiratory tract has been omitted in the interests of brevity. Includes tables, charts, and a bibliography containing reports unavailable to reviewers as well as those examined.

MCMASTER, PHILIP D., *et alii*. The inhibition of vesiculation in mustard gas. H. lesions of human skin by BAL, to March, 1945. (OSRD Report 5027.) Off. Pub. Bd., Report, PB 5877. 1945. 33 pp. Price: Microfilm, 50c.; Photostat, $3.00.

The observation was confirmed that vesiculation of mustard gas lesions can be inhibited by prolonged applications of combinations of liquid BAL and BAL ointments. The experiments gave no evidence that the effect of BAL in preventing vesiculation of H lesions was specific. On the contrary, BAL, when applied to skin wet with H, spread and trapped the vesicant and rendered the lesions worse. There was no apparent neutralizing effect. BAL ointment did not inhibit vesiculation in lesions produced by heat or by tincture of cantharides. Tables and photographs are included. This is a final report under contract with the Rockefeller Institute for Medical Research.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on May 23, 1946, at the Royal Prince Alfred Hospital. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital.

Carcinoma Treated by Radon.

DR. P. D. BRADDON showed two patients suffering from carcinoma who had been treated by radon. The first patient was a male, aged fifty-three years, who had been treated by radon for a columnar-celled carcinoma of the rectum, and when shown, twenty months afterwards, was completely symptomless and had no clinical signs of carcinoma either local or remote.[1]

The second patient had had an extensive intraanal and perianal carcinoma treated by a two-plane radon implant, and when shown, over two years later, was completely free of all signs of disease.

Maldevelopment of the Female Genital Tract.

DR. MURIEL B. MCILRATH showed a female patient, aged eighteen years, who when she reported on October 10, 1944, had never menstruated. She suffered from headaches at monthly intervals which lasted for one or two days, but had no monthly attacks of pain. The bowels were open regularly, and she had no urinary symptoms. She was treated with "Antuitrin S" and stilbœstrol for some months without effect.

On examination, the patient was seen to be a stout, healthy girl, with well-developed breasts. No abnormality was detected in the heart or chest. The pubic hair was scanty, the *labia majora* were normal, the *labia minora* were poorly developed, and the urethra was in the normal situation. There was no vaginal orifice at all. Rectal examination revealed a cystic mass the size of a football, which was palpable in front of the rectum.

On December 8, 1944, with the patient under general anæsthesia and in the lithotomy position, a catheter was passed into the bladder. An incision was made behind the urethra and a plane of cleavage was found extending upward for about half an inch. At the upper end the cystic swelling could be felt. The wound was packed and the patient was put in the Trendelenburg position. The abdomen was opened by a mid-line incision, and old menstrual blood was found in the abdominal cavity. Double hæmatosalpinx and hæmatometra were present, and the uterus was lying on top of the cystic mass palpated *per rectum* and through the perineal incision. The ovaries were normal. The left Fallopian tube was so grossly damaged that it was removed; the appendix contained many fæcoliths and was also removed. The abdominal wound was closed and the patient was put in the lithotomy position. The cystic swelling was incised and the old menstrual blood was drained away. A drainage tube was inserted up to the cervix and stitched into position. It was removed five days later, and dilators were passed every day. Dr. McIlrath said that the patient had made an uninterrupted recovery and had menstruated normally ever since. Dilatation had been necessary to keep the band of fibrous tissue patent. It could be seen lying half an inch from the vaginal introitus. The case was one in which the vaginal bulbs had failed to open into the urogenital sinus.

Dr. McIlrath's second patient was a woman, aged twenty-one years, who on April 13, 1944, had been married for eight weeks. Penetration was impossible. The menstrual periods occurred every twenty-two days and lasted for four days, and were always painful. There was no discharge. The bowels were open regularly, and she had no urinary symptoms. She had had no previous illnesses. A right inguinal hernia had been repaired two years previously.

On examination, the patient was seen to be small and well developed. No abnormality was detected in the heart and chest. A herniotomy scar was present on the right side. The urine was acid and contained no abnormal constituents. Two vaginæ were found and what seemed to be two cervices. The passages were so small that adequate pelvic examination was impossible.

On May 1, 1944, with the patient under ether anæsthesia (open method) and in the lithotomy position, the right vagina was explored, and the cervix was found and dilated. Iodotol was injected and an X-ray photograph was taken. The left vagina was explored and the cervix was found; but an attempt to dilate the os failed, as the vagina was too small to allow necessary manipulation. The septum was crushed with enterotribes and divided, and both cervices were inspected. The left cervix was now dilated, and a probe was passed into the left uterus, which was found to lie behind and to the left of the right uterus. Iodotol was injected into the left uterus and an X-ray photograph was taken. Both uteri appeared to be well enough developed to bear a pregnancy. On May 29 the vagina was healed. Some mild cervicitis was present, for which suppositories were prescribed. On June 19 the cervices were healthy.

On December 21, 1945, the patient reported that her last menstrual period had occurred on October 22, and that she had suffered from nausea for two weeks. Her blood pressure was 120 millimetres of mercury (systolic) and 70 millimetres (diastolic). The urine was acid and contained no abnormal constituents. Both uteri were enlarged and the cervices were softened. It appeared that the right uterus was pregnant. The pelvic measurements were: interspinous, nine inches; intercristal, ten inches; external conjugate, 7·5 inches. The outlet was narrowed. The expected date of confinement was August 3, 1946.

On January 23, 1946, the left uterus was found to be the seat of a three months' pregnancy. The patient's blood pressure was 120 millimetres of mercury (systolic) and 70 millimetres (diastolic). The urine was normal. Dr. McIlrath said that the case was one of pregnancy occurring in a *uterus pseudodidelphys*. The pregnancy had proceeded smoothly.

(To be continued.)

[1] This case has already been reported in THE MEDICAL JOURNAL OF AUSTRALIA of April 6, 1946, at page 477.

NOTICE.

THE first general meeting of the Section of Clinical Pathology of the Victorian Branch of the British Medical Association will be held in the Medical Society Hall, 426, Albert Street, East Melbourne, at 8.15 o'clock p.m. on Friday, November 29, 1946. The business is as follows:

1. The proposed constitution of the section.

2. Discussion on the desirability of the establishment of a diploma of clinical pathology at the University of Melbourne.

3. Address by guest speaker Dr. B. K. Rank on "Aspects of Clinical Pathology Related to Skin Grafting". This will include discussion on bacteriology and problems associated with homologous and heterologous grafts and methods of ensuring adhesion of grafts.

Membership of the section is open to all members of the Branch. Other professional laboratory workers who are interested in the subject under discussion are notified that invitations will be extended to them on application to the director of their laboratory (if a member of the section) or to the honorary secretary, Section of Clinical Pathology, British Medical Association (Victorian Branch), 426, Albert Street, East Melbourne, C.2.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioner has been released from full-time duty with His Majesty's Forces and has resumed civil practice as from the date mentioned:

Dr. P. T. Millard, 69, Baylis Street, Wagga Wagga, New South Wales (June 4, 1946).

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on June 12, 1946, at the Children's Hospital, Melbourne, DR. A. P. DERHAM, the President, in the chair. Parts of this report appeared in the issues of November 2 and 9, 1946.

Pemphigus.

DR. CLIFFORD SAWREY showed a male child, aged three years, of whose illness he thought pemphigus seemed the most

likely diagnosis. The boy had been admitted to the Launceston General Hospital in April, 1945, suffering from a widespread skin disease of obscure origin. This had been present for at least three months, during which period the child had received penicillin injections and local treatment without success at Spencer Hospital, Wynyard, Tasmania. Apparently the skin lesion had developed first on his hands, where blister-like eruptions had formed with excoriation around the blisters. Subsequently these blisters had become generalized on the arms, face, trunk and legs. The blebs at first were irritable, and secondary infection occurred through scratching. Early in the course of the patient's disease the skin blebs had practically disappeared for a few days, but had recurred in a generalized form. During the period at the Launceston General Hospital the temperature was intermittently elevated. The child was regarded as suffering from pemphigus and treated with calamine lotion and 1% gentian violet cream, as well as with penicillin given by the intramuscular route.

Dr. Sawrey said that the child was admitted to the Children's Hospital, Melbourne, on May 7, 1946. His skin was the site of generalized bullous lesions involving even the face and eyelids, varying in size from tiny pustules to bullæ an inch in diameter. The contents varied from serous fluid to pus. The intervening skin was normal. The bullæ did not appear to be itchy. Culture of the aspirated contents of the skin blebs produced a profuse growth of *Staphylococcus aureus* and hæmolytic streptococci. When the sensitivity of these organisms to penicillin was tested, it was found that the *Streptococcus hæmolyticus* was inhibited by 0·0039 unit of penicillin per millilitre and the *Staphylococcus aureus* by 4·0 units per millilitre. Examination of the child's blood gave the following information: the hæmoglobin value was 80%, the erythrocytes numbered 3,890 per cubic millimetre and the leucocytes 34,600 per cubic millimetre; 10% of the leucocytes were eosinophile cells. The Kline and Wassermann tests failed to produce reactions. The temperature was slightly raised, and general examination otherwise revealed no particular abnormality. Dr. Sawrey said that the disease had resisted all forms of treatment, local and general, including intensive penicillin therapy, after which there was a temporary improvement only. Local applications included "Monacrin" (1/2,000), potassium permanganate (1/10,000), mercury perchloride and ultra-violet irradiation.

DR. ARTHUR DAY thanked Dr. Sawrey for presenting the patient on his behalf. He said that though pemphigus was rarely seen, it was the most likely diagnosis. The long history with exacerbations favoured that view, as did the absence of erythema surrounding the lesions. The child's general condition was unusually good for this disease, although when he arrived in Melbourne he looked ill. The acute type of pemphigus ran a rapidly fatal course of a few weeks' duration. In the case of the child under discussion, the term *"pemphigus vulgaris"* might be applied. This was a more chronic disorder. In the differential diagnosis *dermatitis herpetiformis* had to be considered. In this, however, the lesions were multiform—erythematous, papular, vesicular and bullous. All types might occur at the same time, though at one examination most of them might be papular. On another occasion, vesicular lesions might predominate, or bullous lesions might be more striking. The disease occurred at all ages. The lesions of *dermatitis herpetiformis* in children tended to be more generalized and less grouped than in adults. Irritation was pronounced. Against this diagnosis were the lack of irritation and the absence of any evidence of grouping of the lesions. Dr. Day said that bullous impetigo had suggested itself as the diagnosis in the early stages of the disease, but he believed the demonstration of staphylococci and streptococci by cultural methods favoured secondary contamination. There was no evidence of local contagion by contiguous lesions. *Erythema multiforme* occurred symmetrically on the limbs as a rule. Bullous syphilide was seen more frequently on the palms and soles of a younger child. In conclusion, Dr. Day said that he had seen drug rashes from phenobarbitone and iodide resembling that on the child presented, but he still thought pemphigus was the most likely diagnosis.

DR. ROBERT SOUTHBY remarked on the good general condition of the child after a seventeen months' illness. He had noticed that some of the lesions appeared to be irritable and that fresh lesions were appearing. Sensitivity to one of the vitamin or drug preparations was possibly a factor. Dr. Southby suggested that all treatment be suspended for a month and that the child be shown at a subsequent meeting.

DR. MOSTYN POWELL suggested that blood chemistry investigations might shed some light on the diagnosis.

DR. KATE CAMPBELL said that the possibility of an allergic basis for the complaint had occurred to her. The eosinophilia suggested this, if worms could be excluded.

DR. A. P. DERHAM said that the condition might well be treated as an allergic phenomenon, the child being kept on a basal diet and ephedrine being administered.

Dr. Day, in reply, said that vitamins had not been "pushed" and the lesions had not been irritable. A group of bullous lesions might appear without any urticarial manifestations whilst the child was being dressed. All internal treatment had been suspended for a period with only slight and temporary improvement.

Sudeck's Post-Traumatic Bone Atrophy.

DR. BRUCE HALLOWS said that he had shown his patient, a girl, aged fourteen years, suffering from Sudeck's disease, at the October, 1945, meeting of the society. Considerable doubt was at that time thrown upon the diagnosis, and various alternative suggestions were put forward. These included infective osteitis, simple disuse atrophy, fractured neck of the talus, and *os trigonum*. Dr. Hallows said that the latest X-ray films taken in May, 1946, had been studied by Dr. Colin Macdonald, and had presented indubitable proof that what was considered to be an *os trigonum* was in reality a small fracture of the posterior tip of the talus. The most recent films showed that this fracture was now firmly united with the body of the talus. Since October, 1945, when radiological signs of Sudeck's atrophy were apparent, the bones had lost the "spotty" atrophic appearance previously observed and were now of normal density. Moreover, the associated vascular phenomena had completely disappeared. The patient had resumed normal activity.

DR. MOSTYN POWELL said that his only experience in this type of case had been in Palestine. A soldier had been sent from Greece with the story that he had been frost-bitten. The foot and calf were blue and œdematous. There was no evidence of frost bite of the toes. The X-ray film showed a peculiar picture as in Dr. Hallows's case. It remained for a surgical colleague to establish the diagnosis. The limb was put in a spica bandage, and overnight the œdema disappeared and the foot returned to normal. The only explanation that could be offered was that a constriction bandage had been tied around the leg causing Sudeck's atrophy.

DR. ERIC PRICE said that Dr. Powell's story was full of merit, but should not be left uncapped by the presumption that the treatment carried out was correct for Sudeck's atrophy. Sudeck's atrophy was considered to be an extension of the syndrome of disuse; but a feature in Sudeck's atrophy was the intensity of the changes and their persistence and perhaps the degree of pain. In one case in which he had operated for another complaint and in which these changes developed, penicillin had effected a cure. Treatment was to use the affected limb, and the less surgical interference the better.

Dr. Hallows, in reply, said that vascular changes were amongst the criteria of diagnosis. These had been present in this case. De Takats and Miller had attributed these changes to reflex vasodilatation, which might occur through an axone reflex or through a spinal cord reflex via efferent dilators in the posterior roots or through vasodilators in the sympathetic system.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 200, of October 24, 1946.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

The appointments of the following officers have been terminated: Temporary Surgeon Lieutenants (D) Edwin Alan Mobbs, 12th August, 1946, Alan David Willington, 16th August, 1946, and Gordon Spence Hewlett, 5th September, 1946.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

The appointments of the following officers have been terminated: Acting Surgeon Lieutenant-Commander Donald

Ross Macaulay Cameron, 31st July, 1946, Surgeon Lieutenants Stewart Horton Delbridge Preston, 12th August, 1946, Leonard Hugh Catchlove, 19th August, 1946, and Henry Bertram Holmes, 21st August, 1946.

AUSTRALIAN MILITARY FORCES.

Colonel (Honorary Brigadier) W. P. MacCallum, D.S.O., M.C., Reserve of Officers (A.A.M.C.), is appointed Honorary Physician to His Royal Highness the Governor-General of Australia, 1st June, 1946, *vice* Colonel (Honorary Brigadier) W. W. S. Johnston, C.B.E., D.S.O., M.C., E.D., Reserve of Officers (A.A.M.C.), who relinquished the appointment, 31st May, 1946.

Colonel (Honorary Brigadier) J. Steigrad, C.B.E., V.D., Reserve of Officers (A.A.M.C.), is appointed Honorary Surgeon to His Royal Highness the Governor-General of Australia, 1st June, 1946, *vice* Colonel K. B. Fraser, E.D., Reserve of Officers (A.A.M.C.), who relinquished the appointment, 31st May, 1946.—(Ex. Min. No. 197—Approved 23rd October, 1946.)

Australian Army Medical Corps.

SX2910 Lieutenant-Colonel W. M. Irwin relinquishes command of 121st Australian General Hospital (Australian Imperial Force) and is placed upon the Regimental Supernumerary List, 8th August, 1946.

TX2140 Captain (Temporary Major) J. W. H. Merry is appointed to command 20th Australian Field Ambulance, 26th March, 1946.

VX14845 Lieutenant-Colonel N. M. Eadie is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform, 24th July, 1946.

106th Australian General Hospital.—SX34532 Captain M. K. Smith is removed from the Regimental Supernumerary List, 1st August, 1946.

No. 112 (*Brisbane*) *Military Hospital*: *To be Temporary Major, 26th July,* 1946.—QX59681 Captain G. G. Macdonald.

VX48626 Lieutenant-Colonel A. C. Mendelsohn is transferred to the Reserve of Officers (Australian Army Medical Corps), 17th August, 1946.

N78718 Lieutenant-Colonel N. M. Gibson, O.B.E., is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform, 27th August, 1946.

SX21586 Lieutenant-Colonel C. B. Sangster relinquishes command of No. 105 (Adelaide) Military Hospital and is placed upon the Regimental Supernumerary List, 16th August, 1946.

NX102551 (NP10081) Major J. R. Nimmo is appointed to command No. 105 (Adelaide) Military Hospital, 16th August, 1946.

NX139073 Lieutenant-Colonel (Temporary Colonel) J. Leah, C.B.E., relinquishes the rank of Temporary Colonel, is transferred to the Reserve of Officers (Australian Army Medical Corps) with the rank of Lieutenant-Colonel, and is granted the honorary rank of Colonel, 2nd August, 1946.

NX130933 Major (Temporary Lieutenant-Colonel) T. W. Freeman relinquishes the rank of Temporary Lieutenant-Colonel and is transferred to the Reserve of Officers (Australian Army Medical Corps), 1st August, 1946.

To be Temporary Lieutenant-Colonel, 16th August, 1946.—TX2140 Captain (Temporary Major) J. W. H. Merry.

Reserve of Officers.

The undermentioned officers are transferred to the Reserve of Officers with effect from the dates indicated, and, on the date prior to such transfer, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of transfer to the Reserve of Officers:

QX44129 Captain (Temporary Major) N. B. Wilmer, 16th August, 1946.

2nd/11th Australian General Hospital.—NX115475 Major A. Owen, 16th August, 1946.

101st Australian General Hospital (*Australian Imperial Force*).—NX113195 Major J. P. E. O'Brien, 9th August, 1946, Captains NX201329 L. W. Middleton, 26th July, 1946, NX130807 A. G. Moffitt, 9th August, 1946, and NX122978 R. V. W. Roberts, 17th August, 1946.

No. 102 (*Holland Park*) *Military Hospital.*—VX95080 Captain N. Mendelson, 14th August, 1946.

No. 110 (*Perth*) *Military Hospital.*—Captains WX40069 J. A. Scott and SX28215 L. E. Verco, 15th August, 1946.

No. 112 (*Brisbane*) *Military Hospital.*—QX59646 Captain R. A. McCullagh, 17th August, 1946.

No. 113 (*Concord*) *Military Hospital.*—NX76363 Major H. B. Gatenby, 20th August, 1946, and NX133393 Captain J. C. Church, 10th August, 1946.

No. 115 (*Heidelberg*) *Military Hospital.*—VX98459 Captain (Temporary Major) J. M. J. Jens, 22nd August, 1946, and VX91416 Captain J. B. F. Tucker, 15th August, 1946.

128th Australian General Hospital (*Australian Imperial Force*).—NX203798 Captain B. C. Eglitzky, 16th August, 1946.

70th Australian Camp Hospital.—NX201243 Captain J. P. Swain, 9th August, 1946.

80th Australian Camp Hospital.—VX95142 Captain R. G. Fox, 16th August, 1946.

103rd Australian Convalescent Depot.—NX203561 Captain D. C. Pope, 15th August, 1946.

Inter-Service Medical Wing Demobilization Centres (*Australian Military Forces Component*).—Captains NX166031 P. A. Hardcastle, 10th August, 1946, NX204415 F. Rosenfield, 16th August, 1946, VX138562 J. A. P. Buchanan, 20th August, 1946, VX64739 D. P. Churton and VX91888 A. R. Steel, 17th August, 1946, and SX32595 J. S. McK. Stewart, 28th August, 1946.

Captains NX70188 E. M. Cortis, 13th August, 1946, NX208031 B. W. G. Cameron, 2nd August, 1946, and NX108709 M. L. Verso, 6th August, 1946.

2nd/2nd Australian General Hospital.—VX114203 Captain I. O. Stahle, 1st August, 1946.

101st Australian General Hospital (*Australian Imperial Force*).—Captains NX77376 A. L. Stephenson, 31st July, 1946, NX126937 H. G. Royle, 30th July, 1946, and NX135425 J. W. Follent, 8th August, 1946.

No. 102 (*Holland Park*) *Military Hospital.*—VX14709 Major H. D. Stewart, 13th August, 1946, and QX53503 Captain G. Pasquarelli, 24th July, 1946.

No. 105 (*Adelaide*) *Military Hospital.*—SX34386 Captain J. A. Lewis, 14th August, 1946.

106th Australian General Hospital.—VX117266 Major A. L. Newson, 6th August, 1946, Captains NX128607 C. W. A. J. Schlink, 27th July, 1946, and VX64811 B. T. Glanville-Hicks and TX6321 W. Rosenthal, 20th July, 1946.

No. 110 (*Perth*) *Military Hospital.*—VX64992 Captain A. D. Meares, 10th August, 1946.

No. 112 (*Brisbane*) *Military Hospital.*—QX55211 Captain K. H. Hooper, 26th July, 1946.

No. 113 (*Concord*) *Military Hospital.*—NX76252 Major C. J. Gibson, 26th July, 1946, VX112205 Captain (Temporary Major) C. L. Goghlan, 6th August, 1946, Captains NFX203862 A. M. Henderson, 31st July, 1946, NX116034 V. H. Cumberland, 2nd August, 1946, and NX77337 D. S. Stuckey, 30th July, 1946.

No. 115 (*Heidelberg*) *Military Hospital.*—VX39070 Major H. B. Kay, 26th July, 1946, Captains VX117062 R. C. Hunt, 7th August, 1946, VX91417 J. C. Zwar, 9th August, 1946, and VX108032 R. H. Symington, 20th July, 1946.

12th Australian Camp Hospital.—NX136178 Captain T. P. G. Bateman, 24th July, 1946.

2nd Australian Field Ambulance (*Australian Imperial Force*).—VX66743 Captain K. Keely, 25th July, 1946.

3rd Australian Ambulance Train (*Australian Imperial Force*).—NX144006 Major J. T. Anderson, 19th July, 1946.

Inter-Service Medical Wing Demobilization Centres (*Australian Military Forces Component*).—Majors VX39105 W. L. Forsyth, 7th August, 1946, and VX39097 J. R. Stawell, 23rd July, 1946, Captains QX59114 O. N. Lloyd, 25th July, 1946, QX36747 J. B. D. Smith, 23rd July, 1946, QX61549 G. L. Wadeson, 24th July, 1946, N429524 R. C. Sork, 19th July, 1946, NX77347 A. J. Mooney and NX167898 A. E. McK. Reddel, 23rd July, 1946, SX19133 D. N. Kekwick, 9th August, 1946, SX20147 R. A. Goodhart, 20th July, 1946, and V148348 M. F. Lauricella, 2nd August, 1946.

The undermentioned officers are transferred to the Reserve of Officers with effect from the dates indicated, and on the date prior to such transfer, where applicable, cease to be seconded. Officers holding temporary rank relinquish such temporary rank on the date of transfer to the Reserve of Officers and are granted from such date honorary rank on the Reserve of Officers equivalent to the temporary rank relinquished:

No. 113 (*Concord*) *Military Hospital.*—NX201299 Captain (Temporary Major) S. W. Wherrett, 9th August, 1946.

No. 115 (*Heidelberg*) *Military Hospital.*—VX92810 Captain (Temporary Major) D. F. Lawson, 14th August, 1946.

103rd Australian Convalescent Depot.—NX152739 Captain (Temporary Major) J. M. Wilshire, 10th August, 1946.

1st Australian Out-Patients' Depot.—NX103304 Captain (Temporary Major) D. C. G. Bracken, 17th August, 1946.

Retired List.

The undermentioned officers are placed upon the Retired List on the dates indicated with permission to retain their present substantive rank and wear the prescribed uniform.

Where applicable they cease to be seconded and relinquish any temporary rank held, with effect from the date of placement upon the Retired List.

No. 113 (Concord) Military Hospital.—NFX135721 Captain H. M. I. Braye, 17th August, 1946.

2nd/2nd Australian Casualty Clearing Station.—NX170034 Captain J. V. Sanders, 20th August, 1946.

106th Australian General Hospital.—NFX116547 Captain E. Cammack (*née* Scott-Young), 20th July, 1946.

107th Australian Convalescent Depot.—V147982 Captain I. V. Yoffa, 24th July, 1946.

2nd/1st Australian Casualty Clearing Station.—VX117222 Captain H. L. McCay, 1st August, 1946.

Inter-Service Medical Wing Demobilization Centres (Australian Military Forces Component).—NX166837 Captain G. R. Cockburn, 20th June, 1946.

Reserve Citizen Military Forces.

1st Military District.—Major A. M. Langan is retired, 19th August, 1946. The following officers are retired 5th July, 1946: Lieutenant-Colonel J. J. Power, D.S.O., Captains C. N. Matheson, M.C., and R. P. Rundle, Honorary Captain M. J. Gallagher, Captains H. S. Walsh and J. H. Blackburn, Honorary Captains P. F. V. Crowe, R. J. Nash, C. D. Barlow, W. A. Beet and I. McLelland.

2nd Military District.—The undermentioned officers are retired: Captain O. J. Ellis, 30th September, 1945, Honorary Major F. G. Griffiths, 6th September, 1945, and Honorary Captain G. T. Hunter, 29th September, 1945.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The appointments of the following Flight Lieutenants are terminated on demobilization with effect from the dates indicated: J. W. Gardiner (257561), G. R. Jones (257478), 2nd September, 1946, J. I. Guenther (287414), 4th September, 1946, J. H. Cloke (257500), W. J. Stevenson (257483), 9th September, 1946, R. A. Godfrey-Smith (257579), 16th September, 1946.

The appointments of the following Temporary Flight Lieutenants are terminated on demobilization with effect from the dates indicated: J. H. P. Abbott (267131), S. G. Barr (267515), G. W. Browne (267689), A. I. Lane (266930), A. J. Lundie (263436), N. J. Michael (267728), 12th September, 1946, I. H. F. Swain (267551), 17th September, 1946.

The appointments of the following Flight Lieutenants are terminated on demobilization with effect from the dates indicated: G. S. Stable (277461), 15th August, 1946, E. L. Bird (267089), 18th September, 1946, W. T. Lesslie (267619), 18th September, 1946, I. C. Morrison (266409), 24th September, 1946, H. H. Martin (257505), 30th September, 1946, K. H. Pike (277438), 1st October, 1946.

Correspondence.

SOME PROBLEMS OF BACKACHE AND SCIATICA.

SIR: Dr. Lindon (THE MEDICAL JOURNAL OF AUSTRALIA, November 2, 1946, page 646) has stated that I have read into certain articles considerably more than the authors themselves have claimed, and, in an attempt to justify this statement, he goes to the opposite extreme and underquotes from one of the articles concerned.

Josey and Murphy described seven cases of cervical disk disease. "And in one case, one case only", writes Dr. Lindon, "they observed that pressure upon the back of the disk produced the precordial pain of angina." Dr. Lindon has omitted to mention that, of the seven cases, three were not operated on and in two more no mention is made as to whether pressure was applied to the disk or not. Such pressure was applied in the remaining two cases, and in each pain was produced, but in one of these the authors were not sure whether the pain was produced by pressure on the disk or by traction on an anæsthetized nerve root. This is a somewhat different state of affairs from that which Dr. Lindon implies.

Dr. Lindon has described the concealed disk and the mobility test as "good examples of wishful thinking" (he bases this statement on a small series as against Dandy's thousand odd cases), but he has ignored my offer of a motion picture film which would demonstrate the test—a strange attitude on the part of one who is candid enough to admit that his pre-operative diagnosis was correct in only 48 out of 72 cases.

I note with regret that Dr. Lindon has not withdrawn his reference to chiropractors. If the mobility test was quite valueless I would still take exception to this remark, as it is discourteous to the memory of a great surgeon, and I hope that Dr. Lindon will close this correspondence by withdrawing it.

Yours, etc.,

JAMES H. YOUNG.

131, Scarborough Beach Road,
Mount Hawthorn,
Western Australia.
November 5, 1946.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

WEEK-END COURSE AT ORANGE.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course will be held at Orange on Saturday and Sunday, December 7 and 8, 1946, in conjunction with the Western Medical Association. The programme of the course, which will be held at the Hotel Canobolas, will be as follows:

Saturday, December 7, 1946.

2 p.m.—Registration.
2.30 p.m.—"Clinical Applications of Electroencephalography: Demonstration of a New Recording Unit", Dr. Gilbert Phillips.
4 p.m.—"The General Principles of Diagnosis and Treatment of Diseases of the Skin", Dr. J. C. Belisario.

Sunday, December 8, 1946.

10 a.m.—"The Management of Head Injuries" and film on local anæsthetic methods in neurosurgery, Dr. Gilbert Phillips.
11.30 a.m.—"Sterility in the Female", Dr. Bruce Hittmann.
2 p.m.—"Treatment of Habitual Abortion and Threatened Abortion", Dr. Bruce Hittmann.
3 p.m.—"Drug Eruptions, with Special Reference to Sulphonamides", Dr. J. C. Belisario.

The fee for the course will be £1 1s., except for members of the defence forces, who may attend without fee. Those intending to be present are requested to notify Dr. S. R. Dawes, Honorary Secretary, Western Medical Association, 83, Kite Street, Orange.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

A COURSE IN PATHOLOGY.

THE Melbourne Permanent Post-Graduate Committee announces that a course in pathology suitable for candidates for the D.L.O. examination Part II will be conducted by Dr. G. S. Swinburne at the Department of Pathology, University of Melbourne, on Tuesdays at 4 o'clock p.m., commencing on November 19. There will be twelve lectures: November 19 to December 17 and January 21 to March 14.

The fee for this course is eight guineas. Applications from those proposing to attend should be sent to the Post-Graduate Committee, College of Surgeons, Spring Street, C.1, as soon as possible. Those entitled to financial assistance under the part-time Commonwealth Rehabilitation Training Scheme are requested to indicate this fact on their application.

Obituary.

ALBERT WALLACE WEIHEN.

WE regret to announce the death of Dr. Albert Wallace Weihen, which occurred on November 5, 1946, at Darling Point, New South Wales.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Jack, Ian Bruce, M.B., B.S., 1941 (Univ. Sydney), 336, Beamish Street, Campsie.

Gronsfeld, Willy, M.B., B.S., 1946 (Univ. Sydney), "Telrail", 21A, Bondi Road, Bondi Junction.

Goldberg, Heinrich Marcel, M.B., B.S., 1946 (Univ. Sydney), 22, Stafford Street, Double Bay.

THE FEDERAL MEDICAL WAR RELIEF FUND.

THE following contributions to the Federal Medical War Relief Fund have been received:

New South Wales.

The Ophthalmic Association Limited, £367 6s.

Eastern Suburbs Medical Association—War Practice Protection Fund, £195.

H. F. Wilson, £26 5s.

V. R. Elphick, £21.

C. D. Bateman, K. H. Broome, E. McA. Steel, £10 10s.

C. F. A. de Monchaux, K. S. Harrison, K. Klein, J. McVittie, E. H. M. Stephen, N. R. Wyndham, £5 5s.

S. D. Meares (second contribution), £4 4s.

Total: £676 15s.

Grand total: £15,980 14s. 6d.

Books Received.

"Operative Surgery", by George Bankoff, M.D., D.Ch., F.R.F.P.S., F.R.C.S.E.; 1946. London: Medical Publications Limited. 8½" × 5½", pp. 416, with many illustrations. Price: 63s.

"Tuberculosis in the West Indies: Report on Sociological and Clinical Survey", by W. Santon Gilmour, M.B.; 1946. London: The National Association for the Prevention of Tuberculosis. 8½" × 5½", pp. 222, with illustrations.

"The Principles and Practice of War Surgery, with Special Reference to the Biological Method of Treatment of Wounds and Fractures", by J. Trueta, M.D., Hon.D.Sc. (Oxon.); Third Edition; 1946. London: Hamish Hamilton Medical Books, in conjunction with William Heinemann (Medical Books) Limited. 9½" × 6", pp. 444, with many illustrations. Price: 42s.

"Aids to Dermatology", by Robert M. B. Mackenna, M.A., M.D., B.Ch. (Camb.), F.R.C.P. (London); Third Edition; 1946. London: Baillière, Tindall and Cox. 6¼" × 4", pp. 318. Price: 6s.

"Aids to Public Health", by Llywelyn Roberts, M.D. (Hygiene), M.R.C.P., D.P.H.; Fifth Edition; 1946. London: Baillière, Tindall and Cox. 6¼" × 4", pp. 269. Price: 6s.

"A Guide for the Tuberculous Patient", by G. S. Erwin, M.D.; Second Edition; 1946. London: William Heinemann (Medical Books) Limited. 6¼" × 4", pp. 128. Price: 3s. 6d.

Medical Appointments.

Dr. J. S. Stewart has been appointed assistant medical superintendent, Royal Adelaide Hospital, Adelaide.

Professor A. A. Abbie and Professor J. B. Cleland have been appointed honorary physical anthropologist and honorary botanist respectively to the Museum Department, South Australia.

Dr. D. J. Thomas has been appointed representative of the Victorian Branch of the British Medical Association and Dr. C. A. M. Renou has been appointed representative of the Royal Australasian College of Surgeons on the Anti-Cancer Council of Victoria.

Dr. H. C. Stone has been appointed Medical Superintendent of the Mental Hospital, Beechworth, Victoria.

Dr. M. G. Sarre has been appointed Resuscitation Registrar at the Royal Adelaide Hospital, Adelaide.

Dr. A. Fryberg has been appointed government representative on the Senate of the University of Queensland, in terms of Section 14 of *The University of Queensland Acts*, 1909 to 1941.

Dr. E. P. Cherry, Dr. W. M. Irwin and Dr. P. D. Goatcher have been appointed registrars at the Royal Adelaide Hospital, Adelaide.

Dr. R. A. Goodhart has been appointed an honorary medical officer at the Wallaroo Hospital, South Australia.

Diary for the Month.

Nov. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 20.—Western Australian Branch, B.M.A.: General Meeting.
Nov. 21.—Victorian Branch, B.M.A.: Executive Meeting.
Nov. 21.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 22.—Queensland Branch, B.M.A.: Council Meeting.
Nov. 26.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 27.—Victorian Branch, B.M.A.: Council Meeting.
Nov. 28.—South Australian Branch, B.M.A.: Council Meeting.
Nov. 28.—New South Wales Branch, B.M.A.: Branch Meeting.
Dec. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Dec. 4.—Victorian Branch, B.M.A.: Branch Meeting.
Dec. 4.—Victorian Branch, B.M.A.: Council Meeting.
Dec. 4.—Western Australian Branch, B.M.A.: Council Meeting.
Dec. 5.—New South Wales Branch, B.M.A.: Special Groups Committee.
Dec. 9.—Victorian Branch, B.M.A.: Executive Committee.
Dec. 10.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
Dec. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Dec. 11.—Victorian Branch, B.M.A.: Council Meeting.
Dec. 12.—New South Wales Branch, B.M.A.: Branch Meeting.
Dec. 12.—South Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

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